

DISEASES TRANSMITTED BY FOODS

(A CLASSIFICATION AND SUMMARY)

SECOND EDITION

**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES
PUBLIC HEALTH SERVICE
CENTERS FOR DISEASE CONTROL
CENTER FOR PROFESSIONAL DEVELOPMENT AND TRAINING
ATLANTA, GEORGIA 30333**

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INTRODUCTION

This reference summarizes the following data: etiologic agents and their nature, sources and important reservoirs, epidemiology, foods frequently involved in outbreaks, specimens and samples to take in outbreak investigations, laboratory approaches, and general control measures of foodborne diseases that have been reported throughout the world. This information is organized for rapid review and comparison of the same kinds of data about different diseases.

Diseases transmitted by foods are frequently classified either as poisonings or infections. Poisonings are caused by ingesting toxicants that are found in tissues of certain plants and animals, metabolic products (toxins) formed and excreted by microorganisms (such as bacteria, fungi, or algae) while they multiply in foods, or poisonous substances that may be intentionally or incidentally added to foods as a result of producing, processing, transporting, or storing. Infections are caused by the entrance of pathogenic microorganisms into the body and the reaction of body tissues to their presence or to the toxins they generate within the body. Intestinal infections may be manifested by *in vivo* enterotoxin production or mucosal penetration. After mucosal penetration, the organisms multiply in the mucosa or pass into other tissues. This classification is illustrated on the adjacent page.

In this reference, the foodborne diseases are classified on the basis of the type of agent responsible for the illness--bacterial, viral and rickettsial, parasitic (protozoan, cestodes, nematodes, trematodes, helminths), fungal (mycotoxin or mushroom), poisonous plants, toxic animals, poisonous chemicals, and radionuclides. In each category, the diseases of contemporary importance are listed first. The relative importance of each disease does, however, vary from time to time and from place to place. This reference also includes diseases that have been reported as being foodborne even though proof is lacking. It also includes diseases in which the causative agent has been found in foods but transmission through foods is unknown, and enteric diseases in which the transmission through foods is possible. No attempt has been made to discuss all poisonous plants, poisonous chemicals, or radionuclides. Only those plants that are used as food or mistaken for food and those that illustrate a different class of poison are reviewed. There has been no attempt to list all foods that have been incriminated as vehicles in outbreaks. Except in the cases of rare diseases, only foods of primary public health importance are listed.

The symptoms of each disease are usually in the order either of their occurrence or of their predominance. Individual cases, however, will not manifest all the symptoms and, in some cases, additional signs and symptoms will occur. The incubation period (latent period in the case of poisonings) of a disease is the time from ingestion of the contaminated food until the first symptoms appear; individual cases may have incubation periods that vary from the stated times. Control measures are listed in order of their relative importance. A complete bibliography of the literature from which this summary was compiled would be extensive, so only one or a few specific references for each disease are given, along with review articles and texts in each category.

Blank spaces in the text indicate that the appropriate information is insignificant, is unknown, or has not come to the attention of the author. The author encourages comment on missing diseases and information and submission of additional data for revision of the summary.

DISEASES TRANSMITTED BY FOODS (1-14) 1

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
BACTERIAL DISEASES (1-25) 1						
Diseases of Contemporary Importance						
SAFOMONELLOSIS						
Salmonellosis (8,11,16,111, 123,136,183, 239,275) ¹	<i>Salmonella</i> <i>choleraesuis</i> , <i>S. enteritidis</i> serotypes <i>Typhimurium</i> , <i>Heidelberg</i> , <i>Derby</i> , <i>Java</i> , <i>Infantis</i> , <i>Agona</i> , <i>Enteritidis</i> , <i>Montevideo</i> , <i>Newport</i> , <i>Panama</i> , <i>Stanley</i> , etc.	Gram-negative, non-spore forming, (mostly motile) rod. Aerobic, facultatively anaerobic. Possesses O (somatic) and two phases of H (flagellar) anti- gens. Usually more than 10^5 required to cause illness. Over 1600 serotypes known, but only about 50 commonly occur.	Feces of infected domestic or wild animals and man. Infants, aged and malnourished per- sons and those with concomitant diseases are more susceptible. Carrier state usually lasts a few days to a few weeks, but some- times for months. Fifty percent of infected persons carry <i>salmonellae</i> for 2-4 weeks. Occasionally waterborne.	Meat, poultry and eggs and their products. Other incrimi- nated foods have included coconut, yeast, cotton- seed protein, smoked fish, dry milk, chocolate candy.	Feces (stools, fecal swabs, filter paper wipes). Suspect foods; environ- mental swabs (if serotypes). Pre- selective enrich- ment, plating, screening, sero- typing. Phage- typing for <i>Typhimurium</i> , <i>Panama</i> , <i>Enteri-</i> <i>tidis</i> , <i>Infantis</i> , and <i>Thompson</i> .	Chill foods rapidly in small quantities. Cook foods thoroughly. Pasteurize egg prod- ucts and milk. Avoid cross-contamination from raw to cooked foods. Wash hands after touching raw meat. Sanitize equip- ment. Heat-treat feed and feed ingre- dients. Process meat and poultry in san- itary manner. Maintain farm sanitation. Practice personal hygiene. Protect food and feed from animal, human, bird, insect, and rodent excreta.
Typhoid Fever (Enteric fever) (111,112, 176,239, 275)	<i>Salmonella</i> <i>typhi</i>	Similar to other <i>salmonellae</i> but adapted to human host. Possesses Vi (capsular) antigens as well as O and H anti- gens. Usually more than 10^5 required to cause illness.	7 to 28 days, mean 14 days. In foodborne outbreaks, may be shorter incubation period.	High protein foods, milk, shellfish. Cooked foods that have been handled and then eaten without fur- ther heat treatment.	Feces, urine, bile, gall- stones, blood (during early course of ill- ness), bone marrow. Sus- pect food, sewer swabs. Laboratory as above plus phage typing. Demonstration of specific O agglutinins in blood or titer rise in conva- lescent serum.	Practice personal hy- giene. Restrict carriers from handling food. Chill foods rapidly in small quanti- ties. Cook foods thoroughly. Pasteur- ize milk. Age cheese 90 days. Harvest shellfish from un- polluted waters. Prepare and process food in sanitary manner. Protect and treat water. Dispose of sewage in sanitary manner. Immunize. Chloramphenicol ther- apy. Cholecystectomy frequently ends the carrier state.

¹ Numbers within () refer to references.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Paratyphoid Fever (Enteric fever) (111,239, 275)	Salmonella enteritidis serotypes Paratyphi A, Paratyphi B, Paratyphi C, Sendai, and any serotype other than S. typhi) that invades the blood stream and causes septicemia.	Similar to other salmonellae but more or less adapted to human host or capable of reaching blood stream.	7 to 15 days. Blood stream infection. Headache, profuse perspiration, nausea, vomiting, abdominal pain, enlarged spleen, diarrhea, sometimes rose spots. Similar but sometimes milder and of shorter duration (1 to 3 weeks) than typhoid fever.	Feces and urine of infected persons. Carriers sometimes involved in transmission.	Meat, poultry, eggs, milk, and their products. Other foods contaminated by sewage.	Feces, urine, blood. Suspect foods.	Chill foods rapidly in small quantities. Practice personal hygiene. Cook foods thoroughly. Pasteurize milk and eggs. Protect and treat water; Harvest shellfish from unpolluted waters; dispose of sewage in sanitary manner. Vaccine of questionable value in conferring immunity.
Arizona Infection (110,135)	Arizona pneumoniae	Gram-negative, non-sporeforming motile rod.	2 to 46 hours, usually 12 hours.	Eggs, turkey, chicken, cream-filled pastry, ice cream, custard containing eggs.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Avoid cross contamination from raw to cooked food. Clean and sanitize equipment. Reheat leftover food thoroughly.	
Staphylococcal Intoxication (Staphylococcus enterotoxigenic) Staphylococcus food poisoning)	Toxins D,E, or F Staphylococcus aureus. Toxins elaborated in foods.	Gram-positive, non-sporeforming, non-motile cocci occurring in irregular, grape-like clusters. Aerobic, facultatively anaerobic; coagulase-positive; ferments mannitol; grows well in 10% salt media; produces lipase and hemolysin; often produces orange or yellow pigments.	1 to 7 hours, usually 2 to 4 hours. Sudden onset of nausea, excessive salivation, vomiting, retching, diarrhea, abdominal cramps, dehydration, sweating, weakness, prostration. Fever usually does not occur. Short duration of not more than a day or two.	Nose and throat discharges; hands and skin; infected cuts, wounds, burns; sauces and boils; pimples; acne; feces. Anterior nares of man are the primary reservoirs. Mastitic udders of cows and ewes. Arthritic and bruised tissues of poultry. Foods are usually contaminated after cooking by persons cutting, slicing, chopping or otherwise handling them and then kept at room temperature several hours or stored in large containers.	Cooked ham, meat products;poultry, from ill. Nasal swab, pus from infected sores from food workers. Suspect food.	Vomitus, feces from ill. Nasal swab, pus from infected sores from food workers. Suspect food.	Chill foods rapidly in small quantities. Prepare foods the day of serving, whenever possible. Restrict illness (diarrhea, colds, infected cuts) from food workers. Sanitize equipment. Thorough cooking, reheating, pasteurizing destroys the organism but not the toxin.
	(94,109, 119,208)	Frequently lysed by phage type group III. Resistant to many antibiotics. Toxin is protein (18 amino acids), heat stable. Less than 1 ug can cause illness.			Organism: Selective enumeration, isolation, lipase (egg yolk) reaction, coagulase test, phagotyping.	Toxin: Extraction, concentration, gel diffusion.	

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
<i>Clostridium perfringens</i> Gastro-enteritis (A. Large numbers of 172,165, 280)	<i>Clostridium perfringens</i> (welchii) type A. Large numbers of 172,165, 280) cells must be ingested.	Gram-positive, sporeforming, non-motile rod. Aerobic. Produces lecithinase. Strains form either heat-resistant (some survive boiling for 1 to 5 hours) or heat-sensitive spores. Heating encourages spores to germinate. Approximately 90 known serotypes. Usually more than 10^6 required to cause illness.	8 to 24 hours, median 12 hours. Acute abdominal pain, diarrhea. Dehydration and prostration. Nausea, vomiting, fever and chills are rare. Short duration of 1 day or less.	Feces of infected persons and animals. Soil, dust, sewage. Both raw and cooked foods are frequently contaminated with <i>C. perfringens</i> .	Cooked meat or poultry, gravy, stew, and meat pies.	Feces, suspect food; environmental swabs (if serotype).	Chill food rapidly in small quantities. Prepare foods the day of serving whenever possible. Use clean pans for storage. Hold hot foods at 140F or above. Practice personal hygiene. Cure meats adequately. Disposal of sewage in sanitary manner. Thorough cooking will destroy vegetative cells but not heat-resistant spores. Reheat leftover food to 160F or above.
<i>Clostridium perfringens</i> (Barnhard) (216)	<i>Clostridium perfringens</i> type C (formerly type F). Necrotoxin released during growth in gut.	Gram-positive, sporeforming, non-motile rod. Aerobic. Produces lecithinase and necrotoxin. Strains differ in minor antigens.	6 hours to 6 days, usually 24 hours. Diarrhea, prolonged abdominal pain, gangrene of small intestine, shock, toxemia. Case fatality rate: 40%.	Animal feces. Malnutrition and diet may predispose people to attack. Cooked meat held without refrigeration for many hours. Only two reported outbreaks.	Pork, other meat, fish.	Feces, bowel contents, blood, suspect food.	Eat balanced diet. Chill foods rapidly in small quantities. Thorough cooking will destroy vegetative cells but not heat-resistant spores. Reheat leftover food to >160F. Hold hot food at >140 F.
<i>Enteritis Necroticans</i> (Pig Bel, Darmbrand) (151,155)	<i>Clostridium perfringens</i> type C (formerly type F). Necrotoxin elaborated in foods.	Large, Gram-positive, spore-forming, motile rod, frequently forms chains. Produces lecithinase. Diarrheagenic thermolabile toxin (133°F for 20 min.)	1/2 to 5 hours. Nausea and vomiting (similar to staphylococcal intoxication), sometimes diarrhea. Short duration of 1 day or less.	Soil and dust. Foods are usually kept at room temperature, held warm (but not hot) for several hours, or stored in large containers in refrigerators.	Boiled rice and fried rice. Custards, cereal products, puddings, sauces, vegetable dishes and soups, meat loaf.	Feces, vomitus. Suspect foods.	Chill foods rapidly in small quantities. Hold hot foods at 140F or above. Avoid room temperature storage. Process and prepare food in sanitary manner. Reheat leftover food to 160F. Use clean pans for storage.
<i>Bacillus cereus</i> Gastro-enteritis (151,155)	<i>Bacillus cereus</i> diarrheagenic exo-enterotoxins of <i>Bacillus cereus</i> . Toxins elaborated in foods.	Emetic thermolabile toxin (survive 259°F for 90 min.). Probably very large numbers (>10 ⁶) required to cause illness.	8 to 16 hours. Nausea, abdominal cramps, watery diarrhea. Short incubation of 1 day or less.				

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Botulism (174, 206, 236, 258, 274)	Toxins A, B, E, or F of <i>Clostridium botulinum</i> . Toxins C and D cause botulism in animals. Type G has not yet caused any human cases. Toxin elaborated in foods, wounds or infant gut.	Gram-positive, spore-forming, motile rod. Anaerobic. Produces neurotoxins that interfere with acetylcholine at peripheria; nerve endings. Spores are among the most heat resistant. Toxins are simple proteins and are heat labile.	2 hours to 6 days, usually 12 to 36 hours. Nausea, vomiting, abdominal pain, and diarrhea may appear early. Headache, vertigo or dizziness, lassitude, double vision, loss of reflex to light, dysphagia, dyspnoea, ataxia, dry mouth, weakness, constipation, respiratory distress, respiratory paralysis. Partial paralysis may persist 6 to 8 months. Sensorium usually clear. Case fatality rate: 35 to 65%. Fatal 3 to 10 days.	Soil, mud, water, intestinal tract of animals. Spores widely distributed in soil, but type varies with location.	Improperly canned low-acid food (green beans, corn, beets, asparagus, chilis, peppers, mushrooms, spinach, figs, olives, tuna). Smoked fish. Fermented foods (seal flippers, salmon eggs). Food stored in vacuum-packed food useful in diagnosis.	Blood serum, feces, stomach contents, autopsy tissue. Sustained food. Food: (Toxin) - extraction, mouse neutralization. Serum and feces: House neutralization of salt. Add sufficient nitrite to pasteurized meat products. Discard swollen cans. Bivalent A-B and monovalent E antitoxins and polyvalent A-B-E and A-B-C-D-E-F antitoxin available for treatment.	Heat containers of low-acid food at high temperatures under pressure for sufficient time. Cook home-canned food thoroughly (boil and stir for 15 min.). Acidify food. Keep food refrigerated. Cure in sufficient concentration.
Campylobacteriosis (<i>Campylo- bacter jejuni</i>)	<i>Campylobacter jejuni</i> (<i>Vibrio fetus</i>)	Gram negative, motile, comma-shaped organism. Forms spirals. Micro-aerophilic.	1 to 7 days, usually 3 to 5 days. Diarrhea (stools often foul smelling, bile stained, watery or mucoid or bloody), abdominal pain, fever, anorexia, malaise, headache, myalgia, nausea, vomiting, arthralgias. Duration 1 to 5 days.	Intestine, liver, and gallbladder of cattle, sheep, pigs, poultry (?), poultry, and other water. Contact animals. Contact with infected animals or their tissues another mode of transmission. Waterborne outbreaks documented.	Raw milk, raw beef liver and suspect food.	Blood, feces, suspect food.	Cook meat thoroughly. Pasteurize milk. Chill foods rapidly in small quantities, avoid cross contamination from raw foods of animal origin.
Scombrotoxin (Histamine Poisoning) (65, 83)	<i>Scombrotoxin</i> , histamine, and probably related or synergistic substance(s), or potentiating conditions.	Histidine in flesh decarboxylated by action of <i>Proteus spp.</i> or other bacteria. Thermostable (boiling for 1 hour).	Few minutes to 1 hour. Metallic, sharp, or peppery taste. Intense headache, dizziness, nausea, vomiting, facial swelling and flushing, epigastric pain, throbbing of carotid and temporal vessels, rapid and weak pulse, burning of throat, thirst, difficulty in swallowing, edema, itching of skin, audible wheezing, diarrhea. Generalized erythema and urticarial eruptions over entire body. Recovery within 12 hours.	Histamine in single volunteer trial.	Flesh of scombroidae fish (tuna, bonito, mackerel, skipjack). Blue dolphin (mahi mahi).	Vomitus, feces, scombroidae fish.	Bacterial isolation and identification. Extraction, separation, colorimetry; fluorometry; intramuscular injection of guinea pigs and ileum loops. Fluorometric assay.
	Histamine precursor or antilog. Enterobacteriaceae, particularly <i>Proteus</i> , and pseudomonads, <i>clostridia</i> , <i>streptococci</i> , and others.	Histamine dilator.					

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
<i>Vibrio</i> parahaemolyticus	<i>Vibrio para-haemolyticus</i>	Gram-negative, straight or curved, motile rod.	2 to 48 hours, usually 12 hours. Abdominal pain, diarrhea (watery stools containing mucus), usually nausea and vomiting, mild fever, chills, headache, prostration. Recovery required to cause illness.	Sea water and marine life. Most illnesses have been reported in Japan and in the warmer months.	Raw foods of marine origin. Saltwater fish, mollusks, crustaceans, and fish products. Cucumbers and salty foods have been implicated (following cross contamination)	Feces. Suspect	Cook seafood thoroughly. Chill food rapidly in small quantities. Prevent cross-contamination from raw sea-foods. Sanitize equipment. Avoid using sea water for rinsing food to be eaten raw or for cleaning.
Cholera (16, 88, 150, 232, 242, 261)	<i>Vibrio cholerae</i> and <i>V. cholerae</i> biotype El Tor, either Inaba or Ogawa serotypes. Enterotoxin (exotoxin) elaborated in small intestine.	Gram-negative, motile, curved rod. Aerobic. High alkalinity (9-9.6) growth tolerance. El Tor biotype is hemolytic. Heat-labile enterotoxin mediates movement of water and ions from tissues into lumen of bowel and results in outpouring of isotonic fluid. Unless gastric acid decreased or neutralized, 10^6 required to cause illness.	2 to 3 days. Sudden onset, profuse watery diarrhea containing mucus (rice-watery stools); abdominal pain, rapid dehydration and collapse, cold and clammy skin, drawn and withered face and hands, intense thirst, hoarseness, faintness, muscular cramps in extremities. Appearance of victim: sunken eyes, prominent cheekbones, washerwoman hands, skin turgor. 50 to 75% can die without proper treatment; <1% if given oral or intravenous rehydration.	Can be found in sea-water probably from human sewage. Fish and shellfish can serve as reservoir. Feces and vomitus of infected humans, persons incubating the disease, and convalescents. Persons with impaired gastric acid production and malnourished persons more susceptible.	Raw mussels, shrimp, fish, cucumbers, possibly raw vegetables, mixed and moist foods. Foods made up of or washed or sprinkled with contaminated water; food prepared in utensils rinsed in contaminated water; foods that receive no further heating; foods handled by infected persons ("hors d'oeuvres").	Feces, vomitus, serum, suspect food, water.	Dispose of sewage in sanitary manner. Protect and treat water. Practice personal hygiene. Cook foods thoroughly. Chill foods rapidly in small quantities. Test for specific agglutinins in blood.
Non-O1 <i>vibrio</i> Gastro-enteritis (98)	Exotoxin of non-O1 vibrios (<i>Vibrio enteritidis</i>) (See <i>V. fluvialis</i> , <i>V. hollisae</i> , and <i>V. mimicus</i>).	Gram-negative, curved rod, identical or similar to <i>V. cholerae</i> but has different antigenic structure.	2 to 3 days. Watery diarrhea, abdominal cramps, nausea, vomiting, dehydration, anuria. Varies from loose stools to cholera-like disease.	Pickled herring, chopped egg on asparagus, shellfish.	Pieces, suspect food.	Alkaline enrichment, plating, identification, agglutination.	Dispose of sewage in sanitary manner. Protect and treat water. Practice personal hygiene. Cook foods thoroughly. Chill food rapidly in small quantities.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Bongkrek Poisoning (110,277)	Toxins (toxoflavin and bongkrekic acid) of <i>Pseudomonas cocovenenans</i>	Gram-negative rod. Depending on culture medium, grows in different cell and colony forms and colony color. Produces heat-stable, unsaturated, fatty acid (bongkrek acid) that interferes with carbohydrate metabolism.	Hyperglycemia followed by hypoglycemia (low glucose level in blood), severe spasms, unconsciousness, and death. High case fatality rate.	<i>P. cocovenenans</i> overgrows Rhizopus mold responsible for fermentation only when the mold grows very poorly. The well-fermented product has never been shown to be toxic. Reported only in Central Indonesia.	Poorly fermented bongkrek (pressed or grated coconut).	Blood. Suspect poorly fermented coconut. Isolation and identification.	Warn local population against ingestion of poorly fermented bongkrek. Lower pH of product.
Shigellosis (Bacillary Dysentery) (16,110, 134,285)	<i>Shigella sonnei</i> , <i>S. flexneri</i> , <i>S. dysenteriae</i> , <i>S. boydii</i>	Gram-negative, non-motile rod. Aerobic, facultatively anaerobic. Similar to <i>Escherichia coli</i> but does not ferment lactose. Relatively fragile. More than 30 serotypes. As few as 10 <i>S. dysenteriae</i> and 100 <i>S. flexneri</i> have caused illness in human volunteers.	Usually Transmitted by Other Means, but Sometimes Foodborne	1 to 7 days, usually less than 4 days. Extremely variable, mild to severe symptoms: Abdominal cramps, fever, chills, diarrhea, watery stools (frequently containing blood, mucus, or pus), tenesmus, lassitude, prostration, nausea, vomiting, dehydration.	Feces of infected persons. Main mode of transmission: person-to-person. Also waterborne. Carriers shed organism for few weeks to 2 months or longer.	Moist, mixed food. Potato, tuna, shrimp, turkey, and macaroni salads, milk, beans, apple cider, and poi reported	Feces, suspect food. Enrichment (foods), selective isolation, bio-chemical identification, serotyping of groups other than <i>Sonnei</i> .

DISEASE	EPILOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
<i>Escherichia coli</i> ² Diarrheas (8,16,110, 240)	Gram-negative, non- sporeforming rod. Both enterotoxigenic and invasive strains cause illness. Both heat- stable and heat-labile enterotoxins produced.	8 to 24 hours, mean 11 hours (invasive type). Fever, chills, headache, myalgia, abdominal cramps, profuse watery diarrhea. Similar to shigellosis. 8 to 44 hours, mean 26 hours (enteotoxigenic type). Diarrhea (rice-water stools), vomiting, dehydration, shock. Usually more than 10^6 required to cause illness.	Feces of infected persons. Possible modes of transmission: Person-to-person, airborne and waterborne. Infants are more susceptible. Possibly important cause travellers' diarrhea.	Cheese, coffee substitute, salmon (?).	Feces, throat swabs, blood from ill, suspect food.	Chill foods rapidly in small quantities. Cook and reheat foods thoroughly. Practice personal hygiene. Prepare foods in sanitary manner. Protect and treat water. Dispose of sewage in sanitary manner.	
Beta-hemolytic Streptococcal Infections (Scarlet Fever, Septic Sore Throat) (106,110, 171)	Gram-positive, non-motile, microaerophilic cocc in chains. Produces beta hemolysis (wide zone of complete hemolysis around colonies on blood agar). Lansfield's Group A and G streptococci. Over 40 antigenic types within Group A.	1 to 3 days. Sore and red throat, painful swallowing, tonsillitis, high fever, headache, nausea, vomiting, malaise, rhinorrhea. Occasionally a rash occurs.	Infected persons. Nose, throat, and lesion discharges. Main mode of transmission: Airborne.	Milk, ice cream, eggs, steamed lobster, potato shrimp, tuna, and egg salads, custard and pudding. Foods usually contain eggs or milk.	Nasal and throat swabs, pus, sputum from workers. Suspect food; environmental swabs (if serotype). Isolation, hemolytic (blood agar) reaction, serotyping.	Chill foods rapidly in small quantities. Practice personal hygiene. Cook foods thoroughly. Pasteurize milk. Exclude workers suffering from respiratory illness or skin lesions from handling food.	

²Serogroups that have caused the invasive-type illness are 025, 028, 0112, 0124, 0136, 0143, 0144, 0147, and 0512; those that have been shown to elaborate enterotoxins are 006, 015, 018, 020, 027, 044, 055, 078, 086, 0111, 0114, 0119, 0125, 0126, 0127, 0128, 0142, 0146, 0148, 0154, 0155, and 0156.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL, MEASURES
Yersiniosis (<i>Yersinia enterocolitica</i> , <i>Y. enterocolitica</i> , <i>Y. pseudotuberculosis</i>)	<i>Yersinia enterocolitica</i> , Coccoid forms predominate in young cultures. Aerobic, facultatively anaerobic. Psychrotrophic. About 10 ⁹ caused illness in volunteers.	Gram-negative, motile rod. Coccoid forms suggesting acute appendicitis, fever, headache, malaise, anorexia, diarrhea, vomiting, nausea, chills, pharyngitis, leukocytosis, erythema nodosum.	24 to 36 hours and longer. Abdominal pain.	Urine and feces of infected animals frequently rodents, dogs, pigs, chickens. Found in soil, dust, and water. Waterborne transmission.	Pork and other meat, raw milk, chocolate milk.	Feces, blood, suspect food, animal tissue, lymph nodes.	Cook foods thoroughly. Protect food from cross contamination. Control rodents. Rapid chilling and refrigeration increases lag and slows growth but may have a selective effect.
Brucellosis (<i>B. melitensis</i> , <i>B. abortus</i> , or <i>B. suis</i>)	<i>Brucella melitensis</i> , non-motile, coccoid to rod-shaped cells. Aerobic, but <i>B. abortus</i> requires CO ₂ .	Gram-negative, encapsulated, non-motile, coccoid to rod-shaped cells. Fever, chills, sweating, insomnia, weakness, malaise, headache, muscle and joint pain, loss of weight, anorexia.	5 to 21 days. May be several months. Insidious onset. Fever, chills, sweating, insomnia, weakness, malaise, headache, muscle and joint pain, loss of weight, anorexia.	Tissue, blood, placenta, urine, milk, vaginal discharge, and aborted fetus of infected animals (cattle, sheep, swine, goats, and horses). Main mode of transmission: Contact with infected tissues.	Raw milk, cheese made of raw goat milk.	Blood, bone marrow, milk, urine, animal tissue (see source).	Eradicate brucellosis from livestock (immunize young animals, restrict movement, test, segregate or slaughter infected animals). Cook food thoroughly. Pasteurize milk and dairy products. Age cheese for at least 90 days.
Tuberculosis (110,144, 205,293)	<i>Mycobacterium tuberculosis</i> and <i>M. bovis</i>	Gram-positive, non-motile, acidfast rod. Aerobic. Contains waxy substance (resistant). Grows slowly (>18-hour generation time).	Variable. Several weeks.	Respiratory secretions of man; milk from diseased cattle. Main mode of transmission: Airborne.	Raw milk.	Sputum, gastric washings, joint fluids, lymph nodes, blood, urine, bone biopsy, suspect food.	Eradicate tuberculosis in animals (test and slaughter reactors). Pasteurize milk. Isolate and treat cases. Immunize with BCG in high prevalence areas.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Nitrite Poisoning (Methemoglobinemia) (141,253)	Nitrite reduced from nitrate by Enterobacteriaceae, staphylococci, <i>Pseudomonas</i> , <i>S. subtilis</i> , <i>C. perfringens</i>	Reduces nitrates to nitrites.	Few hours. Vomiting, cyanosis (blueness of lips and fingers), fall in blood pressure, loss of consciousness.	Excessive nitritation of fields. Occurs in children. Moist foods stored at room temperature.	Spinach	Blood	Do not harvest produce shortly after nitritation. Refrigereate foods. Chill cooked rapidly in shallow containers.
Diphtheria (87,110)	<i>Corynebacterium diphtheriae</i>	Gram-positive, non-motile, pleomorhic rod. Frequently club-shaped swellings possessing metachromatic granules. Cells usually form a palisade. Aerobic, facultatively anaerobic. Toxigenic strains carry a lysogenic phage.	2 to 5 days, sometimes longer. Insidious onset. Inflammation of throat and nose, spreading, grayish exudate of membranous character on uvula, palate, and nostrils. Fever, chills, sore throat, malaise, difficulty swallowing, edema of pharynx, enlarged cervical lymph nodes, prostration. Yellow, blood-stained discharge from nose. Albuminuria and hematuria.	Obligate parasite of man (case, convalescent, carrier). Discharges and secretions from mucous surfaces of nose, pharynx, and nasopharynx. Skin and other lesions of man. Main mode of transmission: Airborne. Milkborne transmission is rare today.	Raw milk, ice cream.	Throat and nose swabs, blood, milk.	Immunize. Pasteurize milk. Prevent contamination by humans after heat treatment of milk. Practice personal hygiene. Isolate cases.
Tularemia (110,244)	<i>Francisella tularensis</i>	Gram-negative, non-motile, pleomorhic rod. Aerobic. Survives quite well at low temperatures. Can penetrate unbroken skin. 10^{10} can cause illness by respiratory or intradermal routes.	8 to 24 hours or longer. Ulcer forms at site of pathogenic invasion. Chills, high fever, prostration, stupor, coma; swollen, tender, suppurative lymph nodes. Course of illness may be fulminant and fatal.	Source: Blood and tissue of infected mammal or infected arthropod. Reservoir: Wild animals, frequently rabbits, wood ticks. Main mode of transmission: Contact with infected tissue. Also transmitted by insect bites and drinking water. Rarely transmitted by food.	Rabbit meat.	Rabbit meat, blood, lymph nodes, muscle and other tissues. Meat.	Cook meat of wild rabbits thoroughly. Use rubber gloves when dressing rabbits. Wear protective clothes and use tick repellent in endemic areas.
						Isolation, agglutination reaction, serology.	Demonstrate specific agglutinins in blood.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Q Fever (Query Fever) (110, 197)	Coxiella <i>(Rickettsia)</i> <i>burnetii</i>	Gram-negative, small, pleomorphic, nonmotile rod, frequently occurring in pairs. Obligate intracellular parasite occurring as clumps and masses within cytoplasm. Markedly resistant to desiccation. Resists 60C for 1 hour.	2 to 4 weeks, mean 20 days. Sudden onset, chills, headache, weakness, malaise, severe sweats, high fever, pneumonia, mild cough, chest pain. Milk are frequent sources. Other sources: Bodies of patients, carcasses, wool, straw, laundry, air. More commonly transmitted by inhalation of aerosols.	Tick (feces), wild animals, cattle, sheep, and goats. Dust and aerosols from animals. Placental tissue, fetal membranes, amniotic fluid and milk are frequent sources. Other sources: Bodies of patients, carcasses, wool, straw, laundry, air. More commonly transmitted by inhalation of aerosols.	Milk (rarely transmitted by this source). Milk, sputum, urine, cerebro-spinal fluid, postmortem tissues, milk.	Pasteurize milk at 145F for 30 minutes or 161F for 15 seconds. Practice personal hygiene (animal workers). Vaccinate animals before shipping to areas having infected animals. Separate pregnant animals for parturition and 3 weeks thereafter. Dispose of placenta and fetal membranes so animals have no access to them.	
Anthrax (Intestinal)	Bacillus <i>anthracis</i>	Gram-positive, non-spore-forming, encapsulated, large rods which frequently form long chains. Aerobic, facultatively anaerobic. Morphologically and biochemically resembles <i>B. cereus</i> .	2 to 3 days. High fever, general weakness, malaise, headache, insomnia, nausea, abdominal pain, vomiting (containing bile and blood) diarrhea; progressing through general toxemia, shock, cyanosis, and death. Gastrointestinal anthrax is frequently fatal.	Tissue, hide, and feces of infected animals. Soil contaminated by infected animals. Main mode of transmission: Contact with infected animals or contaminated hides or materials. Rarely transmitted by food.	Raw or undercooked meat and sausage.	Cook foods thoroughly. Animal postmortem inspection. Isolation of sick animals. Dispose of carcasses to avoid contamination. Vaccinate healthy animals.	
Haverhill Fever (110)	Streptobacillus <i>moniliformis</i>	Gram-negative, non-spore forming, pleomorphic rods forming chains. Tends to fragment	1 to 5 days. Rash. Swollen, red, and painful joints. Sore throat.	Raw milk.	Blood, joint fluid, pus, animal saliva, suspect food.	Pasteurize milk. Control rodents.	

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Disease in Which Proof of Transmission by Foods Is Inconclusive							
Faecal Streptococcal Enteritis (Enterococcal)	Streptococcus faecalis and <i>S. Faecium</i>	Gram-positive cocci in chains. Grows in 6.5% NaCl at pH 9.6 at 50F, at 113F. Withstands 140F for 30 minutes.	2 to 36 hours, usually 6 to 12 hours. Nausea, abdominal pain, diarrhea, sometimes vomiting. Relatively milk and similar to <i>C. Perfringens</i> foodborne illness.	Feces of animals and man.	Sausage, evaporated milk, meat croquettes, meat pies, pudding.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene. Prepare food in sanitary manner.
Gastro-enteritis (110)	Lansfield's Group D streptococci. 10 ³ -10 ⁶ of a few strains of <i>S.</i> faecalis required to cause illness.	Alpha-, beta-, or non-hemolytic.					
Proteus Gastro-enteritis (110,134)	Proteus vulgaris <i>P. mirabilis</i> <i>P. morganii</i> and <i>P. rettgeri</i> (staphylococcal enterotoxin?)	Gram-negative, motile rod. Aerobic, facultatively anaerobic. Produces urease.	3 to 5 hours. Diarrhea, vomiting, abdominal cramps. (Also see scombroid poisoning.)	Feces of animals and man.	Headcheese, ham, cheese, spaghetti. (See scombroid poisoning.)	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.
Providencia Gastro-enteritis (110,134)	Providencia alcalifaciens and <i>P. stuartii</i> (<i>Proteus inconspicua</i>)	Gram-negative, non-motile rod. Aerobic, facultatively anaerobic.	2 to 24 hours. Diarrhea, vomiting, abdominal cramps.	Feces of animals and man.	Chicken.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.
Klebsiella Enteritis (110,134)	Klebsiella pneumoniae, <i>K. ozaenae</i> , and <i>K. rhinoscleromatis</i>	Gram-negative, non-motile, encapsulated rod. Aerobic facultatively anaerobic. Enterotoxin detected.	10 to 15 hours. Headache, dizziness, nausea, abdominal pain, watery stools.	Feces of animals and man. Respiratory tract of man.	Beef, rice.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.
Citrobacter Gastro-enteritis (110,134)	Citrobacter freundii (formerly <i>Escherichia freundii</i>) <i>C. intermedium</i> <i>C. intermedium</i> Bethesda-Ballup Group.	Gram-negative, motile rod. Aerobic, facultatively anaerobic. Citrate positive, colitoxigenes organism. Some antigens same as <i>Salmonella</i> , Arizona, <i>E. coli</i> .	1 to 48 hours, median 12 hours. Diarrhea, abdominal cramps, nausea, vomiting, fever, chills, dizziness.	Feces of animals and man.	Corn pudding, raw milk, macaroni with meat, liver sausage, smoked meat.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.

DISEASE	EPILOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
Enterobacter Gastro-enteritis (110,134)	Enterobacter (Aerobacter) cloacae, <i>E. aerogenes</i> , <i>E. hafniae</i> , and <i>E. liquefaciens</i>	Gram negative, (usually) non-motile rod. Aerobic, facultatively anaerobic. Enterotoxin detected.	2 to 6 hours. Diarrhea, nausea, vomiting, abdominal pain.	Feces of animals and man.	Cream-filled pastry, milk, stew.	Feces, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.
Edwardsiella Enteritidis (110,134, 139)	Edwardsiella tarda	Gram-negative rod. Aerobic, facultatively anaerobic.	Abdominal cramps, diarrhea.	Feces of animals (particularly snakes, other reptiles, scagulls, seals) and man.		Feces, suspect food. Isolation, identification.	Chill foods rapidly in small quantities. Cook foods thoroughly. Practice personal hygiene.
Vibrio vulnificus Septicemia (97,98)	Vibrio vulnificus	Gram-negative, motile, curved rod. Ferments lactose.	<24 hours, median 16. Malaise, chills, fever, prostration, sometimes vomiting and diarrhea, hypotension. Metastatic cutaneous lesions (erythematous or ecchymotic areas on extremities, vesicles formed and necrotic ulcers) within 36 hours. Death frequently occurs.	Sea water. Frequently pre-existing hyponic disease. Wound infections also occur in warmer months.	Raw oysters.	Blood, infected tissues. Isolation, identification.	Cook seafood thoroughly.
Vibrio mimicus Diarrhea (127)	Vibrio mimicus	Gram-negative, motile, vibrio similar to <i>V. cholerae</i> but does not ferment sucrose, oxidase-positive, VP negative.	Diarrhea	Sea water, shellfish, marine crustacea	Shellfish	Feces, suspect food. Isolation, identification, serotyping, antibiograms, enterotoxin assay.	Cook seafood thoroughly. Chill food rapidly in small quantities. Avoid cross-contamination.
Vibrio fluvialis Diarrhea (196)	Vibrio fluvialis (Group EF-6 and F)	Gram-negative motile vibrio similar to <i>aeromonas</i> and vibrios, ferments sucrose.	Diarrhea (frequently containing blood and pus), vomiting, abdominal pain, dehydration, fever.	Sea water, river water, shellfish, and marine crustacea.	Unknown (seafood?)	Feces, suspect food. Isolation, identification, serotyping, antibiograms.	Chill foods rapidly in small quantities. Cook food thoroughly.
Vibrio cholerae Diarrhea (168)	Vibrio cholerae	Gram-negative motile vibrio. Halophilic.	Diarrhea			Feces, suspect food. Isolation, identification.	Chill foods rapidly in small quantities. Cook food thoroughly.

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Pseudomonas aeruginosa Gastro-enteritis (110,131, 294)	Pseudomonas aeruginosa	Gram-negative, motile rod. Aerobic, facultatively anaerobic. Produces Pyocyanin and Fluorescin. Forms blue pus in infections. Highly resistant to most common antimicrobial agents and disinfectants. Heat-labile and heat-stable enterotoxins detected.	Few days. Diarrhea, abdominal cramps, nausea, vomiting, dehydration, cyanosis.	Skin lesions, feces of man, water, sewage, soil, vegetables. Also transmitted via hospital environment.	Milk, rabbit, syrup, human milk,	Feces, urine, pus, suspect food.	Chill foods rapidly in small quantities. Pasteurize milk. Cook food thoroughly. Practice personal hygiene. Avoid cross contamination from raw foods. Thorough cleaning of kitchen equipment.
Aeromonas Enteritis (110,278)	Aeromonas hydrophila, A. salmonicida, A. punctata	Gram-negative rod. Aerobic, facultatively anaerobic. Enterotoxins detected.	Diarrhea, abdominal pain, fever.	Water, frogs, fish, water.	Salt mackerel, fish, water.	Feces, water, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Treat and disinfect water supplies.
Plesiomonas Shigelloides Enteritis (110)	Plesiomonas shigelloides	Gram-negative rod. Aerobic, facultatively anaerobic. Possesses two common antigens with <i>Shigella</i> . Enterotoxins detected.	Diarrhea, abdominal pain, fever.	Water, fish.	Enrichment, plating, separation from enterics by oxidase test, biochemical identification.	Feces, water, suspect food.	Chill foods rapidly in small quantities. Cook foods thoroughly. Treat and disinfect water supplies.
Bacillus subtilis Gastro-enteritis (110,152)	Bacillus subtilis	Gram-positive, sporeforming, motile rod. Aerobic. Sometimes confused with <i>B. cereus</i> because of morphological and biochemical features.	Average 10 hours, some reports; others 15 to 60 minutes.	Soil and decomposing organic matter.	Fish, turkey, sausage rolls, liver sausage, pickled fish.	Feces or vomitus, suspect food, environmental swabs.	Chill foods rapidly in small quantities. Isolation, identification.
Bacillus brevis Gastro-enteritis (152)	Bacillus brevis	Gram-positive, sporeforming rod.	1 to 10 hours.	Soil and air.	Vomitus, feces.	Vomitus, feces.	Chill foods rapidly in small quantities. Isolation, identification.

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<i>Bacillus licheniformis Gastroenteritis</i> (152)	<i>Bacillus licheniformis</i>	Gram-positive, sporeforming rod.	8 to 12 hours. Diarrhea, vomiting, abdominal cramps, prostration.	Soil and air.	Feces, blood, suspect food. Isolation, identification.	Chill foods rapidly in small quantities. Hold hot food at 140°F or above.
<i>Clostridium bifertmentans Diarrhea</i> (110)	<i>Clostridium bifertmentans</i>	Gram-positive, motile, anaerobic rod.	6 to 7 hours. Diarrhea.	Soil. Potato pie.	Feces. Isolation, identification	Chill foods rapidly in small quantities. Reheat foods thoroughly. Hold hot foods at 140°F or above.
<i>Alcaligenes faecalis</i> (E. coli?)	<i>Alcaligenes faecalis</i> (E. coli?)	Gram-negative, motile, aerobic, coccoid rod.	6 to 13 hours, average 21 hours. Abdominal cramps, diarrhea, vomiting, thirst, headache, dehydration, pyrexia, myalgia.	Soil, vegetation. Meat, poultry.	Feces. Isolation, identification	Chill foods rapidly in small quantities. Protect cooked foods from contamination.
<i>Actinomyces Gastro-enteritis</i> (?)	<i>Actinomyces-like organisms- (Staphylococcal Intoxication or Ichthyotoxicism?)</i>	Branched forms that break into small coccoid segments.	1 hour. Diarrhea, vomiting, abdominal cramps, prostration.	Unknown. Only one reported outbreak.	Roe. Isolation, identification	Cook foods thoroughly. Protect food from contamination. Chill food rapidly in small quantities.
<i>Listeriosis</i> (110, 249)	<i>Listeria monocytogenes</i>	Gram-positive, motile rod. Aerobic, micro-aerophilic. Grows well in 10% NaCl media and survives in 20%. Beta hemolytic grows well at 39°F. Survives 176°F for 5 minutes.	Unknown. Probably 4 days to 3 weeks. Fever, headache, nausea, vomiting, moncytosis, meningitis, septicemia, abortion, localized external or internal lesions, pharyngitis.	Tissues, urine, or milk of infected animals.	Milk, possibly milk products (cream, sour milk, cottage cheese), eggs, meat, poultry tissue.	Animal tissue, Cook foods thoroughly. Pasteurize milk.
<i>Acetobacter melanogenus</i> Intoxication (110)	Products produced by <i>Acetobacter melanogenus</i>	Gram-negative, motile or non-motile rod. Strict aerobes that oxidize ethanol to acetic acid.	Abdominal pain, nausea, vomiting, black tarry stools.	Homemade lager. Contaminant of yeast.	Feces, vomitus, suspect food. Isolation and identification.	Control fermentation of beer.

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Unknown Role in Foodborne Transmission (Pathogenic and Isolated From Foods)							
Mycobacteria Infections (110, 117, 293)	<i>Mycobacteria</i> Runyon Group III (Battye- avian-swine group)	Acid-fast rod. Aerobic. Contains waxy substance (resistant). Non- photochromogenic. Resists destruction by conven- tional pasteur- ization.	Variable. Several weeks.	Infected animals, soil (?).	Unknown, possibly milk (?).	Suspect food, blood. Laboratory: See Tuberculosis.	Increase pasteur- ization temperatures.
Erysipeloid (110)	<i>Erysipelothrix</i> <i>insidiosa</i> , <i>E.</i> <i>erysipeloides</i> , and <i>E. rhusi- opathiae</i>	Gram-positive, non- spore-forming rod with tendency to form long filaments. Microaerophilic, facultative. Resistant to salting, pickling, and smoking.	Few hours. Malaise, generalized pruritus, redness, swelling and itching of infected areas, anemia. Septicemia may develop.	Infected animals and fish. Pri- marily an occupa- tional disease of persons handling these animals or their meat.	Possibly poultry, fish, meat, salted pork (?).	Suspect food, biopsy tissue. Isolation, identification.	Cook foods thoroughly Avoid cross contamina- tion. Thorough clear- ing of meat and fish processing equipment.
Leptospirosis	<i>Leptospira</i>	Spirochetes with fine coiling of their primary spirals. Aerobic. Over 150 sero- types.	4 to 19 days, usually 10 days. Fever, headache, chills, malaise, vomiting, muscular pain. Duration 2 to 4 weeks.	Urine and infected tissues of wild and domestic ani- mals. Waterborne.	Possibly milk, meat, ham, kidneys (?).	Animal tissue, blood, urine, water. Isolation, animal inocu- lation, micro- scopy, serology.	Cook foods thoroughly Protect foods from contamination by animal urine.
Pasteurellosis	<i>Pasteurella</i> (8, 236) <i>multocida</i>	Gram-negative, encapsulated, nonmotile, bi- polar, cocco- bacillus. Pleo- morphic. Aerobic, facultatively anaerobic.	Infection of many sites and symptoms depend upon infected site. Septicemic form.	Infected animals and their secre- tions.	Poultry, vege- tables soiled with animal feces (?).	Sputum, pus, cerebrospinal fluid, blood, urine, infected tissue.	Cook foods thoroughly Protect foods from contamination by animal feces.
						Isolation, identification.	

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VIRAL AND RICKETTSIAL DISEASES (3,4,8,11,12,14,26-30)							
			Epidemiological Evidence of Foodborne Transmission				
Hepatitis A	Hepatitis Virus A (Non-B Virus of Infectious Hepatitis) (HAAg)	Not propagated in tissue culture. Isometric, 27nm in diameter. Probably RNA enterovirus that clumps with con- valescent serum from hepatitis A patients. Remains infec- tive after 1/2 hour at 13°F but inactivated at 212°F for 5 min. Survives freezing.	14 to 50 days, usually 25 to 30 days. Systemic infection characterized by con- stitutional and gastro- intestinal manifesta- tions and by injury to liver. Fever, malaise, lassitude, anorexia, nausea, abdominal dis- comfort, bile in urine, jaundice. Severity of illness increases with age. Duration of a few weeks to several months.	Oysters, clams, milk, orange juice, potato salad, cold cuts, frozen straw- berries, glazed doughnuts, whip- ped cream cakes, sand- wiches, mixed vegetable salads.	Urine, acute or convalescent serum, feces	Prevent pollution of shellfish growing areas. Dispose of sewage in sanitary manner. Treat water by coagulation- settling-filtration- chlorination. Prac- tice Personal hygiene. Cook foods thoroughly. Isolate cases for 7 to 10 days after jaun- dice. Clean and sterilize needles, syringes, and other instruments used for parenteral infections. Give Gamma Globulin to contacts.	
			16 to 48 hours. Nausea, vomiting, abdominal pain, diarrhea, low grade fever, chills, malaise, anorexia, headache, myalgia. Duration 24 to 48 hours.	Oysters, cockles. Could be any food contaminated with feces.	Feces, suspect foods.	Practice personal hygiene. Dispose of sewage in sani- tary manner. Pro- tect and treat (tissue cul- ture).	
Norwalk Virus	Norwalk and Norwalk-like agents (Ditching, W., Cockle Hawaii, Montgomery, County agents).	Unclassified (perhaps parvovirus). Bacteria-free illness when serially trans- mitted to volun- teers. Acid (pH 2.7) resis- tant; survives 140°F for 30 min. 25 x 32 nm size.	Illness primarily associ- ated with older children and adults.	Feces. Illness primarily associ- ated with older children and adults.	Serology (RIA), virus isolation (tissue cul- ture).	Immunize against all three types of polio- virus. Cook foods thoroughly. Practice personal hygiene. Dispose of sewage in sanitary manner.	
Polyomyelitis	Poliiovirus (100,140)	Small RNA, cubical, naked Picornavidae virus. Replicates in cytoplasm. Among most stable viruses known. Resistant to low pH. 3 serotypes - I, II, III.	3 to 21 days, commonly 7 to 12 days.	Feces and pharyn- geal secretions of infected persons. Main route of transmission: Person-to-person. Countries with low standards of hygiene and children infected early. With im- proved hygiene infections occur later in life.	Feces, pharyn- geal swabs, spinal fluid. Tissue culture (monkey or human kidney). Monkey inocula- tions. Serology (cross neutral- ization or complement fixation).		

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Bolivian Hemorrhagic Fever (115, 181, 251)	Machupo virus	Arenaviridae, Tacaribe group. RNA core. Replicates in cytoplasm.	10 to 14 days. Malaise; headache; eye, leg, and back pain; fever; sweats; prostration. Exanthems on throat, flanks, and soft palate. Duration 1 to 2 weeks. Reapses occur. 30% case fatality rate.	Urine of Infected rodent (<i>Calomys callosus</i>). Control rodents. Protect food from contamination. Cook foods thoroughly.	Corn and other cereals. Possibly any food contaminated with rodent urine.	Blood, throat swabs, urine. Serology, complement fixation.	Pasteurize milk. Control ticks. Immunize (USSR).
Russian Spring-Summer Encephalitis (Diphasic Milk Fever) (237)	Russian tick-borne virus complex. Russian Spring-Summer Louping-ill group viruses.	Group B Arbovirus. Probably RNA core. Replicates in cytoplasm.	7 to 14 days. Sudden onset, headache, fever, nausea, vomiting, hyperesthesia, photophobia, weakness, delirium, coma, meningencephalitis, flaccid paralysis - particularly of shoulder. Bindle with residue. Disphasic (fever and meningencephalitis) 4 to 10 days after apparent recovery. Three weeks' duration.	Infected ticks. Animals infected by ticks. Also transmitted to man by tick bites. Reported in Russia.	Raw milk from goats or sheep.	Blood, cerebro-spinal fluid, brain tissue of fatal cases.	Animal inoculations, chick embryo culture, serology.
Kuru (148, 149)	Kuru virus.	Unclassified. Causes marked increase of astrocytes and degeneration of neutrons. Survives 165F for 30 minutes.	Several months or years. Unsteadiness of stance gait, tremore, ataxia. Laughter, abnormalities of extraocular movement and mental changes. Death in 3 to 6 months.	Predominantly affects women and children of Fore tribe in New Guinea. Transmitted by ritual cannibalism. Genetic factor may predispose.	Uncooked human brain tissue.	Brain tissue of fatal cases.	Suppress cannibalism.
Creutzfeldt-Jakob Disease (26, 108, 149)	Agent similar to Kuru agent	Unclassified	12 months to years. Dementia, severe visual disturbances, myoclonic jerking. No remission or recovery.	Infected animal brains.	Inadequately cooked sheep, swine brains (?)	Brain tissue of fatal cases.	Brain tissue of fatal cases.

Viral Diseases Which Could Possibly Be Transmitted By Foods, But Proof Is Lacking

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Lymphocytic chorio-meningitis (114)	Virus of lymphocytic chorio-meningitis	Arenaviridae RNA virus that is probably enveloped.	8 to 21 days, 15 to 21 days to meningeal symptoms. Fever, chills, sore throat, cough, headache, vomiting, neck stiffness, photophobia, acute aseptic meningitis. Recovery in a few weeks.	Nasal secretions, urine, feces, and semen of mice (<i>Mus musculus</i>), and hamsters (<i>Mesocricetus auratus</i>) contaminate man's environment. Dust, skin abrasions involved in transmission. Virus may persist in mouse through life. Guinea pigs, monkeys, dogs, swine also infected.	Unknown, could possibly be any food contaminated by mice.	Blood, urine, nasopharynx or cerebrospinal fluid, CNS tissue.	Control rodents. Clean home and work environments (sanitation).
Lassa Fever (114,115, 210)	Lassa virus	Arenaviridae RNA virus	6 to 14 days. Malaise, asthenia, lassitude, headache, unremitting fever, sore throat, muscular aches, abdominal cramps, loss of appetite, nausea, vomiting, diarrhea, pharyngitis, blurred vision, flushing, subcutaneous hemorrhages, puffed face, swollen neck, oliguria, dysuria. Duration 7 to 21 days. Death 30% to 66%.	Rodent (<i>Mastomys natalensis</i>) transmission (?). Contact with rodent or excreta, eating uncooked rodent flesh, food contaminated by rodent excreta, airborne.	Rodent flesh (?), complement fixation, tissue culture, electron microscopy.	Rodent flesh (?), complement fixation, tissue culture, patients.	Control rodents. Strict isolation of patients.
Rotavirus Gastro-enteritis (Infantile Gastro-enteritis) (30,206)	Rotaviruses (Dyovirus; Orbivirus)	Spherical RNA Reoviridae virus, 65 to 75 nm in diameter. Wheel-like appearance. Replicates in cytoplasm. Two serotypes of human viruses.	1 to 3 days. Vomiting, followed by diarrhea (watery green or yellow stool), malaise, fever, abdominal pain, dehydration. Duration 2 to 16 days in infants, 24 hours or less in adults. Death from dehydration or aspiration of vomitus.	Calves, piglets, monkeys, sheep, mice, and other animals carry identical or similar virus. Person-to-person spread is common. Common cause of diarrhea in infants and young children.	Feces, biopsy of duodenal tissue. Serology (RIA, ELISA) Virus isolation (tissue culture).	Pieces, biopsy of duodenal tissue. Prepare food in sanitary manner. Cook foods thoroughly.	Practice personal hygiene. Dispose of sewage in sanitary manner.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
ECHO Virus Infections (26,286)	ECHO (Enteric cyropathogenic human orphan) viruses. types 1-9, 11-27, 29-34.	Small RNA, cubical, naked viruses. Replicates in cytoplasm. Resistant to low pH. relatively stable. 33 serotypes. Type 18 frequently associated with diarrhea. Also, types 11,19,20.	Few days. Diarrhea (greenish, watery). Fever and respiratory symptoms may accompany diarrhea. Also causes febrile illness, asptic meningitis, and paralysis. Duration of diarrhea, 1 to 5 days.	Transient inhabitant of human alimentary tract and found in feces.	Unknown, could be any contaminated food.	Throat swabs, stools, cerebrospinal fluid, feces. Tissue culture (monkey kidney), serology (hemagglutination, complement fixation).	Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.
Coxsackie Infections Herpangina (Summer Gripe)(26, 126)	Coxsackie Group A viruses (including types 2,4,5, 6,8,10,22,24)	Relatively stable, small RNA, cubical, naked viruses. Replicates in cytoplasm. Resistant to low pH. 24 serotypes and 3 subtypes.	3 to 5 days (1 to 14). Fever, lassitude, anorexia, dysphagia, sore throat, stomatitis (papules on soft palate) vomiting, abdominal pain, convulsions. Viruses also cause febrile illness, asptic meningitis, paralysis, common cold, rash, hand-foot-and-mouth disease (stomatitis with exanthems).	Transient inhabitant of human alimentary tract and readily isolated from feces. Also found in nose and throat discharges. Most illnesses are in children in summer months.	Unknown, could be any contaminated food.	Feces, blood, pharyngeal swabs, cerebro-spinal fluid. Mouse inoculations, serology.	Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.
Pleurodynia (Epidemic Myalgia (26,126)	Coxsackie Group B viruses, types 1,2, 3,4,5,6.	Relatively stable, small RNA, cubical, naked viruses. Replicates in cytoplasm. Resistant to low pH. 6 serotypes.	3 to 5 days (1 to 14). Abrupt onset, intermittent fever, myalgia, anorexia, chest and abdominal pain, headache, malaise. Viruses also cause febrile illness, asptic meningitis, systemic infection (myocarditis) with exanthems.	Transient inhabitants of human alimentary tract and readily isolated from feces. Also found in nose and throat discharges. Most illnesses are in children in summer months.	Unknown, could be any contaminated food.	Feces, throat swabs, cerebro-spinal fluid, blood. Tissue culture (monkey kidney) or human amion cells, mouse inoculation.	Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Adenovirus Infections (26, 153, 268)	Adenoviruses	DNA, cubical, naked viruses. 31 serotypes and 1 subtype (28 isolated from man). Relatively stable.	Few days. Diarrhea, fever, vomiting, abdominal pain. Viruses also cause respiratory or eye infections, septic meningitis.	Unknown, could be any contaminated food. Respiratory and person-to-person spread more common.	Electron microscopy. Tissue culture, serology (hemagglutination).	Feces, pharyngeal or eye infections, urine. Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.	
Reovirus Infections (26, 238)	Reoviruses (Respiratory enteric orphan viruses). Reovirus (formerly classified as ECHO 10).	Double-strand RNA, cubic, naked virus. Replicates in cytoplasm. High heat resistance. Three serotypes of the Reovirus group.	2 days or less. Virus isolated from individuals with a wide range of symptoms - respiratory tract diseases, gastrointestinal diseases, nervous system disease.	Feces, nose, throat. Wide host range of man and animals. Respiratory and person-to-person spread more common.	Unknown, could be any contaminated food.	Feces, nasal or throat swabs. Tissue culture (monkey kidney), serology (hemagglutination).	Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.
Hepatitis B (Serum)	Hepatitis B virus B (HBsAG)	Unclassified, double-strand DNA virus, 24 nm in diameter. Survives 4 hours at 140F. Rapidly destroyed by hypochlorite.	88 to 108 days, average 98 days (oral exposure); shorter (65 days average) after parenteral exposure.	Serum, saliva, nasopharyngeal secretions, semen. Long-term carrier state. High rate of infection in homosexuals, infants from mothers at birth. Period of infectivity lasts until HBsAg disappears.	Unknown, possibly any contaminated food.	Serum, urine. Liver function test, serology (HBsAg) of acute serum.	Practice personal hygiene. Dispose of sewage in sanitary manner. Cook foods thoroughly.
Calicivirus Diarrhea (30)	Calicivirus (WESV)	RNA virus, 35-39 nm in diameter.	Diarrhea.	Unknown, virus related to virus that causes vesicular exanthema of swine.		Feces, suspect foods. Serology, tissue culture.	Cook foods thoroughly, prepare foods in sanitary manner.
Coronavirus Diarrhea (30)	Human coronaviruses. Human enteric coronaviruses.	Enveloped RNA virus, 75-160 nm in diameter.	Diarrhea.	Related agents cause diarrhea in calves, dogs, turkeys.	Unknown.	Feces, suspect foods. Serology, tissue culture.	Cook foods thoroughly, prepare foods in sanitary manner.
Astrovirus Diarrhea (30)	Astrovirus	Unclassified, 28 nm in diameter, star-shaped.	Diarrhea	Agent found in stools of ill and well infants.	Unknown.	Feces. Serology.	Cook foods thoroughly, prepare foods in sanitary manner.

DISEASE, ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
PARASITIC DISEASES (3, 4, 8, 12, 14, 31-37)						
Trichinosis (Trichinellosis, Trichinellassis) (32, 37, 157, 201)	Delicate, thread-like roundworm (nematode). Larvae encyst in duodenum. Females invade mucosa of small intestine, larvae travel via blood and lymph, encyst in muscle.	4 to 28 days, usually 9 days. First stage (intestinal invasion): Nausea, vomiting, diarrhea, abdominal pain. Second stage (muscle penetration): Irregular and persistent fever, edema of eyes, profuse sweating, muscular pain, thirst, chills, skin lesions, weakness, prostration, labored breathing. Third stage (tissue repair): Generalized toxemia, myocarditis. High eosinophile blood count.	Always or usually Transmitted by Foods	Pork, bear meat, walrus flesh, dog meat. Frequently home-made raw pork sausage.	Muscle biopsy (gastrocnemius, deltoid), skin test. Diaphragm muscle of swine and bear.	Cook pork thoroughly (until it turns white) to 137°F or above. Freeze and store pork < 6" thick at 5°F for 20 days, -10°F for 10 days, -20°F for 6 days; > 6" thick at 5°F for 30 days, -10°F for 20 days, -20°F for 12 days. Cook garbage for feeding pigs at 212°F for 30 minutes. Cure meat adequately. Eliminate rodents from hog lots.
Taeniasis (Beef or Pork Tapeworm Infections) (32, 33, 37, 228, 280)	Flatworm (cestode). Ingested larvae (<i>Cysticercus Bovis</i>) develop into adult worms in intestines. Adults attach to mucosa of small intestine by their scolexes. Average length 5 meters. Eggs may remain viable for 6 months.	3 to 6 months. Variable, frequently vague or absent. Nervousness, insomnia, hunger pains, anorexia, weight loss, abdominal pain. Digestive disturbances such as nausea, vomiting, colic, and diarrhea sometimes occur.	Human feces containing eggs or proglottids. Immediate source: Flesh of infected cattle. Occurs in East Africa, South and Central America, Asia, Eastern Europe, Southwest U.S.	Beef.	Microscopy (eggs, proglottids).	Dispose of sewage in sanitary manner. Inspect meat. Cook beef thoroughly (>135°F), freeze (15°F, 10 days). Avoid pasturing cattle where human feces or sewage accumulate. Diagnose and treat cases.
<i>Taenia solium</i> (Pork tapeworm)	Flatworm (cestode). Adult worm attaches to mucosa of small intestine. Average length < 3 meters.	3 to 6 months. Variable, frequently vague or absent. Nervousness, insomnia, hunger pains, anorexia, weight loss, abdominal pain. Digestive disturbances such as nausea, vomiting, colic, and diarrhea sometimes occur.	Human feces containing eggs. Immediate source: flesh of infected pigs. Worldwide. Rare in U.S.	Pork.	Microscopy (eggs, proglottids).	Dispose of sewage in sanitary manner. Inspect meat. Cook pork thoroughly, freeze. Keep swine away from areas where human feces or sewage accumulate. Treat cases.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Cysticercosis (228)	<i>Taenia solium</i> larvae, <i>Cysticercus cellulosae</i>	Larval stage. Cysticerci develop in subcutaneous tissues, muscles, and may localize in brain, eyes, heart, central nervous system.	Pain at site of cysticerci development.	Human feces. Auto-infection.	Probably none, but may be any food or water contaminated by human feces containing eggs of the parasite. Vegetables contaminated with night soil are a possibility. Pork may introduce the tapeworm initially.	Serum, biopsy of infected tissue. Microscopy (cysticerci). X-ray, serology.	Practice personal hygiene. Treat cases. Immunize animals.
Diphyllobothriasis (Fish Tapeworm Infection) (32, 33, 39, 280)	Diphyllobothrium latum (Broad or fish tapeworm) D. pacificum	Flatworm (cestode). Adult attaches to mucosa of small intestine. Length is 10 meters or more.	5 to 6 weeks. Symptoms often trivial or absent. Nausea, vomiting, weakness, dizziness, diarrhea or constipation, anemia may occur.	Infective eggs from human feces, dogs, and other fish-eating mammals contaminate water sources. Intermediate host: Copepods. Immediate source: Flesh of infected fish. Occurs in Great Lakes region and Florida.	Raw or partially cooked or inadequately pickled freshwater fish (pike, pickerel).	Feces, fish. Microscopy (eggs).	Cook freshwater fish thoroughly. Dispose of sewage in sanitary manner. Prevent stream pollution. Freeze fish (14F for 24 hours). Treat cases.
Sparganosis (31, 32, 33, 280)	Sparganum of Diphyllobothrium latum and Spirometra mansonioides and other spp.	Tapeworm (cestode). Ribbon-like larvae.	Month or longer. Tender, puffy areas around site of parasite, irritation and migratory swelling.	Cat and dog feces. Intermediate host: Water fleas (cyclops). Source: Water, fish, frogs, snakes.	Tadpoles, snakes, frogs.	Infected human tissue (biopsy), fish. Microscopy (cross section of larva).	Cook foods thoroughly. Abstain from eating raw flesh of infected animals. Avoid using raw vertebrates as poultices. Protect or boil water.
Angiostrongyliaisis (Eosinophilic meningoencephalitis) (82)	<i>Angiostrongylus cantonensis</i>	Roundworm (nemata). Adult worm lives in pulmonary artery of rats and deposits eggs in blood. Larvae hatch from eggs and travel up trachea where they are swallowed an pass in feces.	14 to 16 days. Gastrointestinal upset, encephalitis (headache, stiffness of neck and back, paresesthesia), low grade fever.	Rat feces. Larvae penetrate terrestrial mollusks (snails and slugs) or marine mollusks. Reported in Asia and Pacific Islands. (82).	Raw crab, prawns, garden slugs, land planarian, shrimp, snails. Raw vegetables (82).	Mollusks, rats, serum, autopsy tissue. Microscopy (worms). Serology.	Cook foods thoroughly, freeze (5F). Avoid eating raw, freshwater prawns, raw land mollusks and raw crab. Customs of people play important role in transmission.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Abdominal Angiostrongyliasis (33)	<i>Angiostrongylus costaricensis</i>	Roundworm (nematode). Worms congregate in appendiceal region, may mature and lay fertile eggs.	Abdominal pain, anorexia, vomiting, fever. Distended abdomen, marked leukocytosis. May persist 2 months.	Mesenteric arteries of rats. Slug intermediate host.	Salad, vegetables contaminated by slugs.	Slugs, rats, serum, autopsy tissue.	Cook foods thoroughly, freeze (50°F). Avoid eating slugs.
Anisakiasis (Herring worm disease) (32, 39, 257, 276)	<i>Anisakis spp.</i> Contracaecum spp.	Roundworm (nematode). Highly resistant to brine, easily killed by 140°F and freezing.	4 to 6 hours. Gastric: Sudden stomach pain, nausea, vomiting, eosinophilia in 7 days. Intestinal: Severe lower abdominal pain, nausea, diarrhea, occult blood in stools, leukocytosis, ascites.	Adult worm lives in intestine of fish-eating sea mammals. Larvae found in herring. Reported in Holland, Japan, England, U.S.	Marine fish or squid. Herring (raw, partially cooked, pickled, smoked).	Stools, intestinal tissue, herring (raw, partially cooked, pickled, smoked).	Cook herring thoroughly. Freeze at -4°F within 12 hours and hold for 24 hours. Preserve with high concentrations of NaCl and hold for 10 days. Proper cleaning of fish to be eaten raw.
Fasciolopsis buski (31, 32, 33)	<i>Fasciolopsis buski</i>	Large intestinal fluke (trematode). Adult attaches to intestinal mucosa.	3 months. Diarrhea alternating with constipation, abdominal pain, nausea, vomiting, anorexia, intestinal obstruction may occur, edema of face and abdomen, weakness.	Human, dog, or hog feces containing fluke eggs contaminating fresh water. Intermediate host: snail. Gercariae encyst on water vegetables. Skins of water vegetables are bitten into and peeled off with teeth.	Water chestnuts, water bamboo, water hyacinths, water caltrop, lotus plant root.	Feces, suspect foods.	Avoid hulling or peeling water plants with teeth or lips - use a knife or drop in boiling water. Dry plants. Cook water-grown vegetables thoroughly. Dispose of sewage in sanitary manner. Control snails. Treat patients. Eradicate water caltrop from endemic areas.
Echinostomiasis (31, 33)	<i>Echinostoma revolutum, E. melli, E. cinetrichis, E. macrorchis, E. recurvatum, E. illocanum, and other spp.</i>	Intestinal fluke (trematode). Adult attaches to small intestinal wall.	Several months. Inflammatory reaction at site of attachment to intestinal wall, intestinal colic, diarrhea.	Infecive feces of man, dogs, fowl, rats contaminate fresh water. Immediate source: Snails. Occurs in Orient.	Raw snails and clams. Also limpets, freshwater fish, or tadpoles.	Feces, suspect foods.	Cook snails and of sewage in sanitary manner.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Himasthla infection (31)	Himasthla mehlensi	Intestinal fluke (trematode)	Gastroenteritis.		Raw clams.	Feces, clams. Microscopy.	Harvest shellfish from unpolluted waters. Cook shell-fish.
Clonorchiasis (31, 32, 33, 39, 187)	Clonorchis sinensis (Chinese liver fluke)	Slender hepatic fluke (trematode). Habitat in man is distal biliary passages and pancreatic duct.	Undetermined. Flukes mature in 16 to 25 days. Probably several weeks. First stage: fever, epigastric pain. Second stage: loss of appetite, diarrhea, low grade fever, tenderness over liver, bile duct obstruction. Third stage: cirrhosis, progressive ascites and edema, jaundice.	Infective feces of man, cats, dogs, hogs, or other animals which are hosts of adult flukes contaminate fresh water. Intermediate host: snails. Encyst in muscle of freshwater fish. Occurs in Orient and Eastern Europe.	Raw or partially cooked fresh, dried, salted, or pickled fish (carp and 80 other species).	Feces, bile, fish. Microscopy (eggs), serology.	Cook freshwater fish thoroughly. Dispose of sewage in sanitary manner. Keep sewage out of streams. Control snails.
Heterophyid infections (31, 32, 33)	Heterophyes heterophyes, <i>Stellantchasmus taenatus</i> and other spp., <i>Haploorchis pumilio</i> , and other spp.	Small intestinal fluke (trematode). Attaches to mucosa of upper levels of small intestine. Similar to Clonorchis.	Several weeks. Abdominal pain; diarrhea containing mucus; heart, brain, or spinal cord involvement may follow.	Infective feces of fish-eating birds and mammals contaminate fresh water. Intermediate host: snails. Encyst in fish muscle. Occurs in Orient, Egypt, and Southeast Europe.	Raw, partially cooked, salted or dried freshwater or brackish-water fish (mullet).	Feces, fish. Microscopy (eggs).	Cook fish thoroughly. Prevent stream pollution. Control snails. Dispose of sewage in sanitary manner.
Opisthorchiiasis (31, 32, 33, 39)	<i>Opisthorchis felineus</i> , <i>O. viverrini</i> , <i>C. sinensis</i> .	Hepatic fluke (trematode), resembles <i>C. sinensis</i> .	Several weeks. Cirrhosis of liver resembling Clonochiasis.	Infective feces from humans and piscivorous mammals, containing eggs. Snails ingest eggs. Cercaria penetrate freshwater cyprinoid fish.	Freshwater fish.	Feces, fish. Microscopy (eggs).	Cook freshwater fish thoroughly. Dispose of sewage in sanitary manner. Keep sewage out of streams. Control snails.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Metagonimiasis (31,32,33)	<i>Metagonimus yokogawai</i>	Small intestinal fluke (trematode). Attaches to mucosa of upper levels of small intestines. Similar to <i>Clonorchis</i> .	Several weeks. Abdominal pain; diarrhea containing mucus; heart, brain, or spinal cord involvement may follow.	Infective feces of fish-eating birds and mammals containing fresh water. Intermediate host: snails. Encyst in fish muscle. Occurs in Orient, Egypt, and Southeast Europe.	Raw, partially cooked, salted or dried fresh-water or brackish-water fish (trout).	Feces, fish. Microscopy (eggs).	Cook fish thoroughly. Prevent stream pollution. Control snails. Dispose of sewage in sanitary manner.
Fascioliasis (Sheep, Liver Infection) (31,32,33, 101,128)	<i>Fasciola hepatica</i> and <i>F. gigantica</i>	Large hepatic fluke (trematode). Fluke burrows through intestinal wall to liver.	Several months. Lesions in bile passages, coughing, vomiting, jaundice, abdominal rigidity, diarrhea, irregular fever, profuse sweating, eosinophilia, systemic intoxication.	Infective feces from humans, sheep, cattle, or other herbivorous animals and omnivorous animals, containing eggs, contaminate fresh water. Intermediate host: snails. Cercariae encyst on aquatic vegetables. Occurs in South and Central America, Southern Europe, Middle East, Hawaii.	Aquatic vegetation, water-cress.	Feces, water-cress.	Eradicate infection in sheep and other herbivorous animals. Omnit watercress in salads in endemic areas. Dispose of sewage in sanitary manner. Drain pastures. Prevent stream pollution. Control snails.
Paragonimiasis (31,32,33, 39,292)	<i>Paragonimus westermani</i> (Oriental lung fluke), <i>P. skrjabini</i> , <i>P. heterotremus</i> , <i>P. tuanishanensis</i> , <i>P. africanus</i> , <i>P. chirai</i> , <i>P. iloktsuenensis</i> .	Plump, oval fluke (trematode). Penetrates intestinal wall and reaches lungs.	Long and variable, many months. Cough, hemoptysis. Roentgenographic findings closely simulate those of pulmonary tuberculosis. Migrations and ectopic development in intestine, lymph glands, genitourinary tract, subcutaneous tissue, and brain.	Sputum and feces from man and other carnivores containing eggs, contaminate fresh water. Intermediate host: snails. Cercariae encyst in freshwater crab or crayfish. Occurs mainly in Orient and Pacific Islands.	Raw or partially cooked crab or crayfish.	Feces, sputum, crab, crayfish. Microscopy (eggs), immuno-diagnostic tests (intradermal, complement fixation), X-ray.	Cook crustacea thoroughly. Heat at 131F for 5 minutes. Dispose of sewage in sanitary manner. Prevent stream pollution. Control snails. Mass treatment in endemic areas.

DISEASE	ETOIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ signs and symptoms	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Dicrocoelias (33, 91)	<i>Dicrocoelium dendriticum</i>	Small hepatic fluke (trematode).	7 weeks. Constipation, diarrhea, abdominal pain, enlarged and tender liver.	Infective animal (cattle, sheep) feces, containing eggs. Snails ingest eggs. Larvae leave snails in slime balls in slime balls that infect ants. Ants are ingested by animals or accidentally by man; rarely by dietary preference. Occurs in tropics (rare).	Ants or foods (raw, unwashed vegetables) contaminated by ants (during picnics, camping, etc.).	Feces, sheep liver. Microscopy (eggs).	Wash vegetables. Cook foods thoroughly. Dispose of sewage in sanitary manner. Keep sewage out of streams. Control snails and ants. Protect foods from ants.
Hymenolepidae Diminuta (31)	<i>Hymenolepis diminuta</i> (Rat tape-worm)	Flatworm (cestode). Length is from 20 to 60 cm.	Diarrhea, abdominal pain, and indefinite gastrointestinal complaints.	Feces of rats, mice, man. Intermediate hosts: insects (fleas, cockroaches, beetles, mealworms) ingest eggs.	Grains and cereals.	Feces, beetles. Microscopy (eggs).	Avoid eating insect-contaminated grains and cereals. Inspect grains and cereals. Rodent stoppage; control rodents in grain storage areas. Insect control.
Hymenolepidae Nama (31)	<i>Hymenolepis nama</i>	Flatworm (cestode). Length is from 25-40 mm.	Abdominal pain, diarrhea, anorexia, dizziness, headache, pruritic rash.	Feces of mice and man. Fleas and beetles may serve as intermediate hosts.	Grains (?)	Feces. Microscopy (eggs).	Sanitary disposal of sewage, rodent control. Protection of food from rodent contamination.
Gastro-dicistasis (31)	<i>Gastroduiscoides hominis</i>	Trematode.	Mucous, diarrhea.	Pigs and deer are reservoirs. Snails may be intermediate hosts.	Vegetables (?)	Feces. Microscopy (parasite or eggs).	Sanitary disposal of sewage.
Dioctophyema (248)	<i>Dioctophyema renale</i> (Giant kidney worm)	Roundworm (nematode) Length 14 to 100 mm.	Renal dysfunction or ureteral obstruction.	Urine of infected large fish-eating mammals.	Fish.	Urine. Microscopy (eggs or worm).	Sanitary disposal of sewage, thorough cooking of fish. Drink safe water.
Toxocariasis (Visceral larvae migrants) (33)	<i>Toxocara cati</i>	Prolonged migration of larvae in human tissue.	Fever, malaise, pallor, anorexia, failure to gain weight, muscle and joint pain, nausea, vomiting, convulsions, pruritic rash.	Infective feces of dog or cat reach soil.	Soil contaminated foods.	Blood. Count eosinophils.	Avoid eating dirt, wash vegetables, deworm dogs.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Gnathostomiasis (Creeping Eruption, Larva Migrans) (31, 32)	<i>Gnathostoma spinigerum</i> <i>G. do洛rosi</i> , <i>G. nipponicum</i>	Roundworm (nematode). Extra-Intestinal sites in humans.	Epigastric pain, nausea, vomiting, edema, fever, granulomatous lesions, stationary abscesses. Infection may persist for years.	Adult parasites in gastric tumors of dogs and cats. Eggs pass in feces to water. Intermediate hosts: Water fleas (cyclops). Immediate source: Fish muscle. Third stage larvae migrate to muscle of species eating infected fish, frogs, birds, snakes and remain infective.	Raw, fermented, or partially cooked fresh-water fish; snakes, birds, mammals.	Emerging worms from skin, abscesses, or natural orifices, biopsy.	Cook foods thoroughly.
Intestinal Myiasis (298)	Diptera. <i>Piophila casei</i> (cheese skipper). <i>Musca domestica</i> (common house fly). <i>Stomoxys calcitrans</i> (stable fly) ³	Larvae of flies that oviposit or larviposit in manure, decaying vegetation, or meat. Lesions, damage to mucous membranes, or hemorrhagic infiltrations occur in the intestines due to secretions or injuries inflicted by mouth parts or spines.	Vomiting, diarrhea, abdominal pain, convulsions.	Flies. ³ Larvae of most flies do not continue development in alimentary tract; thus, only cause a pseudomyiasis (do not feed or continue development, just pass through).	Meat, fruit, watercress, cheese, or other contaminated food or water that has been exposed to flies.	Feces.	Practice good sanitation. Protect foods from insect contamination. Control flies.

³Other Diptera reported as causing myiasis include: *Anisopus fenestratus*, *Psychoda sexpunctata*, *Megaselia scalaris*, *Megaselia spiracularis*, *Eristalis tenax* (Drone Fly), *Eristalis arbustorum*, *Tachina fusca*, *Musca stabulans* (False stable fly), *Fannia canicularis* (Lesser house fly), *Fannia scalaris* (European latrine fly), *Fannia manicata*, *Calliphora vicina* (European blue bottle fly), *Calliphora vomitoria* (Red-bearded blue bottle fly), *Calliphora croceipalpis*, *Chrysomya chloropyga* (Green-tailed blow fly), *Sarcophaga haemorrhooidalis* (Red-tailed flesh fly), *Sarcophaga hirtipes*, *Sarcophaga striata*.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Amebiasis (Amebic dysentery) (16, 31-81, 137, 225, 226)	<i>Entamoeba histolytica</i>	Amebic protozoan has four stages (trophozoite, pre-cyst, cyst, meta-cyst). Vegetative stage (trophozoite) is very fragile; cyst stage does not survive drying. After ingestion, intestinal juices render cyst wall permeable and trophozoite emerges. Vegetative stage multiplies in mucosa or lumen of colon. Encysts in the lumen of intestine. Invades mucosa of colon.	Usually Transmitted by Other Means But Sometimes Foodborne	Human feces containing cysts. Main mode of transmission: Personal contact. More common in tropics, mental institutions, and underdeveloped areas.	Raw vegetables and fruits.	Feces, lesion exudates, material aspirated from ulcers.	Practice personal hygiene (food handlers). Cook foods thoroughly. Dispose of sewage in sanitary manner. Protect and treat water. Control flies. Avoid using human excreta as fertilizer (night soil).
Ascaris (31, 32, 33, 212)	<i>Ascaris lumbricoides</i>	Giant roundworm (nematode). Adult lives free in small intestine. Eggs are very resistant to environmental changes, and in warm, humid areas they remain infective for a year or longer.	2 months.	Infective eggs from human feces. Eggs require several days in soil to allow infective larvae to develop.	Raw vegetables and fruits.	Feces. Microscopy (eggs), serologic tests.	Dispose of sewage in sanitary manner. Practice personal hygiene. Cook foods thoroughly. Compost feces used as fertilizer. Anthelmintic medication - mass treatment every 6 months to 1 year in endemic areas.
Trichuriasis (31, 33, 288)	<i>Trichuris trichiura</i> (nematode)	Eggs are very resistant to environmental changes. Attaches to mucosa of cecum, appendix, colon, and rectum.	Long and variable, several months.	Asymptomatic to abdominal discomfort, emaciation, anemia, constipation, loss of appetite, vomiting.	Any soil-contaminated food(?)	Feces. Microscopy (eggs).	Dispose of sewage in sanitary manner. Practice personal hygiene. Cook foods thoroughly. Prevent children from eating dirt.

DISEASE *	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Capillariasis (31, 33, 39)	Capillaria hepatica and C. philippinenensis (Capillary Liver Worm)	Roundworm (nematode) related to the whipworm. Eggs hatch in duodenum; larva enter intestinal wall and migrate to liver. Causes malabsorption due to atrophic changes in intestinal mucosa.	Month or longer. Diarrhea. Acute or subacute hepatitis with eosinophilia and visceral larva migrans. Rarely reported.	Liver (containing embryonated eggs of peccary, monkey, hare, and rodents. Also transmitted by contaminated soil. freshwater fish.	Liver, fresh-water fish.	Liver biopsy or necropsy. Microscopy (eggs).	Avoid eating liver of reservoir hosts. Cook such foods and fish thoroughly. Prevent children from eating dirt.
Enterobiasis (31, 33, 273)	Enterobius vermicularis (Pinworm)	Roundworm (nematode). Free or superficially attached to mucosa of cecum, appendix, and colon.	Several months. Anal itching, local irritation due to scratching, disturbed sleep, irritability. Frequently no symptoms.	Infective eggs from human feces. Main mode of transmission: Personal contact. Crowding is an important factor. Common in children.	Any contaminated raw foods (?)	Feces (scotch tape "swab"). Microscopy (eggs).	Practice personal hygiene. Cook foods thoroughly. Dispose of sewage in sanitary manner.
Trichostomiasis (31, 39, 287)	Trichostongylus orientalis, T. columbiformis, T. vitrinus, and other spp.	Thread-like roundworm (nematode).	Several months. Asymptomatic to Gastrointestinal symptoms.	Infective eggs from animal feces. Close contact with animals important in transmission.	Night-soil contaminated vegetables (?)	Feces. Microscopy (eggs).	Personal hygiene. Cook foods thoroughly.
Echinococcoses	Echinococcus granulosus (Hydatid Disease) Multiceps multiceps	Flatworm (cestode). Hydatid cyst, 3 years. Eggs may survive for long periods in soil.	Several months to variable, depends on site of cyst. Liver, lungs, kidney, pelvis, heart, bones, or central nervous system may be involved.	Feces (containing eggs) or cornvotes (dogs and wolves) infected with adult worms. Intermediate host: larvae occur in sheep, cattle, pigs, camels, moose, deer. Dogs become infected with hydatid cysts from eating raw foods of animal origin (sheep).	Any contaminated raw food.	Serum. Serology, microscopy (scolices or cysts). X-ray.	Control slaughtering so that dogs do not have access to scraps. Control stray dogs. Incinerate or deeply bury dead animals. Deworm domestic dogs. Practice personal hygiene.
Hydatidosis (31, 33, 39, 260, 280)							Main mode of transmission: contact with dogs.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Alveolar Hydatid Disease (31,33)	Echinococcus multilocularis	Flatworm (cestode). Hydatid cyst, 3 to 6 mm long.	Several months to years. Jaundice, ascites, splenomegaly. Frequently fatal.	Feces of foxes, siled dogs, wolves. Maintained in a fox-vole-fox cycle. The disease is transmitted to man by consuming foods contaminated with excreta of Canidae, handling contaminated soil, eating dirt, or contact with infected animals.	Raw fruits and vegetables.	Serum. Serology, microscopy, (scolices or cysts). X-ray.	Control slaughtering so that dogs do not have access to scraps. Control stray dogs. Incinerate or deeply bury dead animals. Deworm domestic dogs. Practice personal hygiene.
Balantidiasis (Balantidial Dysentery) (31,33, 138,295)	Balantidium coli	Large ciliated protozoan, cysts formed. Habitat is mucosa at lower end of large intestine. Ovoid with tapering and trophozoite; spherical or oval cyst.	Unknown. Sometimes a few days. Diarrhea with mucus, blood, pus, constipation. Necrosis and ulceration produced. Symptoms may last 1 to 4 weeks.	Swine, rats, or human feces. Contact with pigs important in transmission.	Pork, raw food.	Feces. Microscopy.	Practice personal hygiene. Cook foods thoroughly. Treat cases. Control flies.
Giardiasis (16,31,33, 138,193, 203)	Giardia lamblia, G. intestinalis	Flagellated protozoan, forms cysts, habitat in small intestine. Pear-shaped trophozoite, oval cyst.	Variable (1 to 6 weeks). Diarrhea, mucous (tatty) stools, abdominal pain and distention, nausea, vomiting, dehydration, fever. Blocks absorption of fats. Frequently no symptoms are produced.	Cyst in human feces. Common in warm climates and in children. Transmission by personal contact and water.	Post-process contaminated salmon, possibly other unheated foods.	Feces, duodenal drainage. Microscopy.	Practice personal hygiene. Cook foods thoroughly. Dispose of sewage in sanitary manner.
Coccidioidosis (Isospora Infection) (132,138)	Isospora bellii, I. natalensis	Intestinal sporozoa (protozoan). Habitat is small intestine. Uses single host for alternate asexual and sexual generations. Ellipsoidal oocyst, oval sporocyst.	Approximately 8 days. Diarrhea, mucus in fecal discharge, abdominal tenderness and distention, nausea, low-grade fever, chills, anorexia, headache. Duration 10 days.	Ripe oocyst in human feces.	Raw foods.	Feces.	Practice personal hygiene. Cook foods thoroughly. Dispose of sewage in sanitary manner.

DISEASE	ETIOLOGIC AGENT	INCUBATION PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Dientamoeba Infection (31)	<i>Dientamoeba fragilis</i>	Small amebic protozoan, nonencysting trophozoite. Lives in mucosa from cecum to rectum.	Variable. Anorexia, nausea, vomiting, low-grade fever, diarrhea (2 to 3 mushy stools per day), epigastric distress.	Human feces.	Raw foods.	Feces. Microscopy. Practice personal hygiene. Cook foods thoroughly. Dispose of sewage in sanitary manner.
Toxoplasmosis (31,32,33, 132,142,177, 281)	<i>Toxoplasma gondii</i>	Crescent-shaped sporozoa (protozoan). Forms cysts. Survives only a short time in extracellular environment.	Unknown. 10 to 13 days in one common-source outbreak. Fever, lymphocytosis, generalized muscle involvement, headache, myalgia, rash.	Source unknown. Mammals (swine, cats, cattle, sheep) and birds are reservoirs. Placental transmission occurs.	Rare hamburgers, raw or rare venison. Cysts found on meat.	Affected tissue, blood, biopsy of lymph nodes. Microscopy. Serologic and skin test, mouse inoculation.
Sarcosporidiosis (31,39,132, 138)	<i>Sarcocystis lindemanni</i> , <i>S. (Isopora) hominis</i> , <i>S. suisomnis</i> , <i>S. bovinis</i>	Protozoan. Elongated, ovoid shape with innumerable rounded and crescent-shaped sporozoites enclosed in a membrane.	Asymptomatic. When found, usually in striated and cardiac muscle during post-mortem examination. Migratory, local swellings at irregular intervals in muscles, accompanied by asthmatic attacks, have been reported.	Sheep, cattle, horses, pigs, muscle fibers.	Raw meat. Microscopy (oocyst).	Cook meat thoroughly. Freeze meat. Trichinoscopy.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
FUNGAL DISEASES (38-52)							
Mycotoxicoses							
Ergotism, Vascular Type (Saint Anthony's Fire) (51,93, 104,160, 200)	Ergot alkaloids from <i>Claviceps</i> purpurea. Toxic alkaloids: Ergotamine, ergotoxins, and ergometrine groups.	Exerts marked vasoconstrictive effect on arterioles. May be tumorous and organic. Organism appears as an enlarged, purple, spur-shaped body among the other seeds - Sclerotium.	1 to 2 hours. Cangrene form: Lassitude, pain in limbs, cold hands progressing to feet, burning sensation, restriction of blood supply to limbs and feet may result in gangrenous necrosis. Convulsive form: Twichings, tonic spasms, hallucinations, convulsions. May persist for 1 to 3 months or longer	Seed grain of rye and other cereals (wheat, barley, oats), soil, air. Uncommon today.	Rye meal or bread.	Stomach contents, liver, rye.	Hold moisture of rye below 10%. Avoid eating moldy rye.
Ergotism, Enteric Type (95)	Toxins from ergot alkaloids from <i>Claviceps</i> <i>Fusiformis</i> of clavine group alkaloids.	Nausea, vomiting, giddiness, somnolence.	Grains of millet.	Wheat, bajra pear millet, occurred in India.	Avoid eating moldy millet. Hold moisture of millet below 10%.		
Alimentary Toxic Aleukia (ATA) (Epidemic Panmyelo- toxicosis) (51,147, 179,180, 200)	Sporofusari- ogenin glycoside and other toxins from <i>Fusarium</i> sporo- trichoides and <i>F. poae</i> . <i>Cladosporium</i> , <i>Alternaria</i> , <i>Penicillium</i> , and <i>Mucor</i> spp.)	Fungi can grow and produce toxin at -2 to -10C, optimum temperature 24C. (Optimum tempera- ture for toxic production 1.5 to 4C.) Requires 200C to destroy toxin. Toxin has destructive action on blood- forming elements of bone marrow. Nonantigenic. Reduces red and white blood cells and platelets.	Few hours. First stage: Burning sensation in mouth, tongue feels stiff, diarrhea, nausea, vomiting, perspiration. Second stage: Quiescent period. Third stage: (2 weeks to 2 months later): Leukopenia, weakness, hemorrhage of skin and mucous membranes, necrotic areas in mouth, throat, and skin; ga- greenous pharyngitis, fever. Case fatality rates of outbreaks from 2 to 80%.	Soil, air. A disease of rural populations occur- ring mostly in the spring. Occurred in Russia. Mold produces mycotoxins during periods of repeated freezing and thawing.	Grains (millet, wheat, oats, barley, rye, buckwheat) that overwinter under snow. Bread.	Grain, blood, urine.	Thresh, wash, and mill grains. Do not allow grains to overwinter in fields. Exclude toxic grains from animal diet. Burn and deep-plow contami- nated fields. Use fungicides on grains.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Aflatoxi-cosis (49, 51, 156, 189)	Aflatoxin B ₁ , B ₂ , G ₁ , G ₂ from <i>Aspergillus</i> <i>flavus</i> - <i>oryzae</i> group.	These fungi are found worldwide and grow on practically any substrate. Carcinogenic to rats, ducks, and trout. Heat stable. Repto toxin.	2 weeks or longer. Low-grade fever, jaundice, scities, and edema of feet. Fatty infiltrations and cirrosis of liver. 27% fatality rate in Indian outbreak.	Widespread in soil and air. Heavy rains and faulty methods of storage of grains. Reported in India, Africa, Taiwan, and Thailand.	Maize, rice, cottonseed meal, Brazil nuts, palm kernels, peanuts, soy beans, corn, wheat, other cereals, and animal feeds.	Feed, nuts, grains. Isolation of fungi, microcopy, extraction, separation and chromatography. Spectrophotometry, animal feeding tests, and skin testing.	Control moisture during storage of grains. Avoid eating moldy grains. Prevent damage of grains during harvesting. Control insects. Fungicidal mold control. Remove contaminated grains or peanuts from processing line. Chemical extraction of toxin.
Liver Cancer (51)	Aflatoxins	As above.	Years. Liver cancer.	Higher incidence in low moist areas where <i>Populus</i> ingests large quantities of moldy foods than in high dry areas of Africa.	Mostly grains.	As above	As above
Acute Cardiac Beriberi (Shoshin-kake) (49, 51)	Toxins of <i>Penicillium citreoviride</i> suspected.	Cardiac toxin, yellow-rice toxin. Deprivation of respiratory center of medulla oblongata.	Retrospective study in Japan.	Precordial distress with palpitations and tachypnea, dyspnea, nausea, vomiting, anguish, pain, restlessness, at times violently manicical, extremities become cold and cyanotic, pupils dilate, unconsciousness.	Polished rice.	Rice. Extract, mouse testing.	Reduce presence of moldy rice reaching market and avoid eating moldy rice.
Kaschin-Beck Disease (Grov Disease) (220)	Toxins from <i>Fusarium sporotrichiella</i>	Mold widespread in soil.	Soil, air. Occurred in Russia, Korea, Sweden, and China.	Mottled grains. Bread.	Grain, blood, urine. Isolation of fungi, microcopy, extraction, animal feeding tests, and skin testing.	Control moisture during storage of grains. Do not make bread out of moldy grain and avoid eating such bread.	

DISEASE	ETOIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
"drunken bread" Poisoning (147)	Toxins from <i>Fusarium graminearum</i> (roseum)	Headache, vertigo, tinnitus, trembling, shaking of extremities, unstable gait. Flushed face, abdominal pain, nausea, diarrhoea. Euphoria, state of confused consciousness. Milder than ATA. Duration 1 to 2 days.	Soil, air. Reported in Russia.	Grain, rye, bread.	Grain, blood, urine.	Isolation of fungi, microscopy, extraction, animal feeding tests, and skin testing.	Control moisture during storage of grains. Do not make bread out of moldy grain and avoid eating such bread.
Akakabi-Byo (Red Mold disease) (49, 51, 241)	Scirpene derivatives from <i>Fusarium nivale</i> and <i>F. graminearum</i>	Mold causes scab disease of grain.	Vomiting, diarrhea, and anorexia.	Wheat flour, barley, oat, rye, rice.	Grain, blood, urine.	Isolation of fungi, microscopy, extraction, separation chromatography.	Control moisture during storage of grains. Avoid eating moldy grain.
Balkan Endemic Nephropathy (Ochratoxicosis) (51, 136, 191, 265)	Ochratoxin A from <i>Aspergillus ochraceus</i>	Hepatotoxins, nephrotoxins.	Anemia, edema, polyuria, impairment of kidney tubular and glomerular function.	Soil and decaying vegetation. Balkan region.	Sorghum grain, corn.	Grains.	Control moisture during storage. Avoid eating moldy grain.
Onyala (Thrombocytopenic Purpura) (266)	Toxins from <i>Pheima sorghina</i>	Hemorrhagic bullae in mouth, thrombocytopenia, haematuria, vascular system hemorrhages.	Soil, air.	Millet and grain sorghum.	Grain.	Chromatography.	Control moisture during storage of grains. Avoid eating moldy grains.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Epidemic Polyurea (Polydypsia Syndrome)	Toxins from <i>Rhizopus nigricans</i>	Toxin is hydrophilic and heat stable.	Insidious onset, frequent urination, anorexia, thirst, weakness, fatigue, nocturia, nausea, vomiting, electrolyte imbalance, blurred vision, photophobia, palpitation, dyspnea, choking sensation, pain and cramps in limbs, giddiness. Duration 35 to 40 days with single meal exposure. Death has occurred.	Soil, air. Common in parts of India.	Pearl millet grain.	Grain, blood, urine. Isolation of fungi, microscopy, extraction, animal feeding tests, and skin testing.	Control moisture during storage. Avoid eating moldy grain.
Poona Disease (219)							
Muco-Mycotoxic Toxins from Mucoraceae thirum (218)			Sporadic vomiting, hemoptysis.	Reported in India.	Wheat, flour.	Grain, vomitus. Isolation of fungus, microscopy.	Control moisture during storage. Avoid eating moldy grain.
Toxic Moldy Rice Disease (49,184)	Toxins from <i>Penicillium</i> , <i>Fusarium</i> , <i>Rhizopus</i> , <i>Aspergillus</i> spp.	Hepatotoxic for rodents and may induce liver tumors. Organism produces yellow pigments causing rice to appear yellow.	Long periods of incubation. Possibly acute gastroenteritis or cirrhosis and hepatoma.	Soil, air. Reported in Japan.	Yellow rice.	Grain, blood, urine. Isolation of fungi, microscopy, extraction, animal feeding tests, and skin testing.	Control moisture during storage. Avoid eating moldy grain.
Moldy Ragi Poisoning (95)	<i>Heterosporium</i>		Vomiting, diarrhea.	Reported in India.	Finger millet (ragi)		Control moisture during storage. Avoid eating moldy millet.
Kodo Millet Poisoning (95)	<i>Phomopsis paspali</i>		20 minutes and longer. Unconsciousness, delirium with violent tremors of voluntary muscles, vomiting, difficulty in swallowing, giddiness, excessive perspiration, inability to speak or swallow. Duration 24 hours.	Reported in India. Millet left in field in rainy weather.	Kodo millet		Control moisture during storage. Avoid eating moldy millet.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Mushrooms							
Cyclopeptide Poisoning (Mushroom poisoning - cell destruction type)	Amanita toxin, Phallotoxins, Amanitoxins, Virosins.	Intracellular poisons. Terminal synthesis of protein (death angel, death cup), and cell death. Hepatotoxic. Hemolytic. Thermostable, stable to drying. Among one of the most lethal toxins known (0.1 mg/kg lethal dose), approximately 1 mushroom cap weighing 50 g.	6 to 24 hours, usually 10 to 14 hours. First phase: Sudden onset, abdominal pain, nausea, violent vomiting, continued and protracted (few days). Bloody or mucoid diarrhea, loss of strength, thirst, desiccation, muscle cramps. Feeble, rapid pulse, apathy, collapse. Second phase: Asymptomatic but cell destruction occurs (hepatic necrosis). Third phase: Jaundice, renal shutdown, cramps, drowsiness, dilation of pupils, stiff neck, twitching of facial muscles. Loss of consciousness, death due to hepatic necrosis and liver and kidney failure. Duration 3 to 10 days. Case fatality rate - 50%.	Mushroom tissue.	Mushrooms: ⁴ <i>Amanita phalloides</i> (death angel, death cup), <i>A. brunnescens</i> , <i>A. bisporigera</i> , <i>A. ocreata</i> , <i>A. suballiacea</i> , <i>A. tenella</i> , <i>A. verna</i> , <i>A. virosa</i> (destroying angel), <i>Conocybe filaris</i> , <i>Galerina autumnalis</i> , <i>G. fasciculata</i> , <i>G. venenata</i> , <i>G. marginata</i> , <i>Lepiota helveola</i>	Intact fresh mushroom, urine, blood, vomitus, stool, gastric aspirate (spores). Mince, extract, paper and thin-layer chromatography.	Avoid eating toxic varieties. Discard in select-ing edible varieties (positive identification.) As a rule cooking or drying does not destroy toxic ingredients of mushrooms. There are no foolproof tests to differentiate between toxic and nontoxic varieties.
Orellanine Poisoning (40, 42, 90)	Orellanine	Protoplastic poison. Polypeptides causes renal failure.	3 to 14 days. Dry mouth, burning lips, intense thirst followed by nausea, vomiting, abdominal pain, constipation or diarrhea, chills, headache. In severe cases - insufficient renal function, polyuria followed by oliguria, anuria, albuminuria, blood in urine, drowsiness, loss of consciousness, convulsions. Case fatality rate 10-20%.	Mushroom tissue.	Mushrooms: <i>Corthinarius orellanus</i>	Mushrooms, urine. Post-mortem examination.	Avoid eating <i>C. orellanus</i> .

⁴ Suspected etiologic agents: *Amanita magnivularis*, *A. verpa*, *A. behemica*, *Lepiota brunneoincarnata*, *L. subincarnata*, *L. castanea*, *L. felina*.

⁵ History of eating mushrooms is important in diagnosis.

⁶ Suspected etiologic agents: *Corthinarius gentilis*, *C. speciosissimus* (*coronaria*, *eximia*).

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Galerina sulciceps Poisoning (42, 90)	Lethal factor.	Short. Stomach spasms, nausea, dizziness, palpitation of heart, dyspnea, local anesthesia, feeling of "pins and needles", unconsciousness, death within 7 to 24 hours.	Mushroom tissue	Mushroom: <i>Gallerina sulciceps</i>	Avoid eating <i>G. sulciceps</i>		
Monomethyl-hydrazine (Gyromitrin Poisoning) (42, 90)	Hemolytic, hepatotoxic (hydrolysis). Toxic to CNS, Gastro-intestinal irritant. Volatile. Soluble in hot water.	2 to 12 hours, usually 6 to 8 hours. Nausea, feeling of fullness, persistent vomiting, watery diarrhea, abdominal cramps, headache, lassitude, muscular cramps, rapid pulse, high fever, dizziness, faintness, loss of coordination. In severe cases, convulsions, coma, death. Case fatality rate - 15%.	Mushroom tissue. Not poisonous to all people.	"False morel" mushrooms; <i>Gyromitra</i> (<i>Helvella</i>) <i>ambigua</i> , <i>brunnea</i> (<i>H. underwoodii</i>), <i>californica</i> , <i>caroliniana</i> , <i>esculenta</i> , <i>fastigata</i> , <i>gigas</i> , <i>infula</i> .	Mushrooms, blood, urine. Distill, pre- cipitate. Identification of mushrooms. Thin layer chromatography, spectrometry.	Mushrooms, blood, urine. Distill, pre- cipitate. Identification of mushrooms. Thin layer chromatography, spectrometry.	Avoid eating false morels. Discriminate in selecting edible varieties (positive identification). Parboiling fruiting bodies for 5 minutes twice, and discarding water may offer some protection. Dry for over 6 months.

⁷Suspected etiologic agents: *Disciotis (Peziza) venosa*, *G. sphaerospora*, *Helvella (Paxina) acetabula*, *H. crispa*, *H. elastica*, *H. lacunosa*, *Morchella* spp., *Peziza babia*, *P. succosa*, *Sarcosphaera crassa*, *Verpa behemica*.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Muscarine Poisoning - (Mushroom Poisoning - Neurological Effects) (41, 42, 48)	Muscarine	Neurotoxin. Stimulates parasympathetic nerve endings and causes glandular secretions (cholinergic effect). Cases respond to Atropine.	15 to 120 minutes, usually within 30 minutes. Rapid onset. Prolonged salivation, perspiration, tearing, peripheral vasodilation, blurred vision, bradycardia, nausea, vomiting, abdominal cramps, copious, watery diarrhea, slow irregular pulse, pupils constricted, muscle spasms, asthmatic breathing, cardiac or respiratory failure (rare). Senorum ordinarily clear. Case fatality rate 6-12%.	Mushroom tissue. Occurs more frequently in May or June.	Mushrooms; ⁸ <i>Amanita muscaria</i> (fly agaric), <i>A. pantherina</i> (panther), Clitocybe, <i>I. hudsonii</i> (Jack O'lantern), <i>C. cerasata</i> , <i>C. dealbata</i> , <i>C. rivulosa</i> , <i>C. truncicola</i> , <i>Inocybe napipes</i> , <i>I. fastigiata</i> , <i>I. geophylla</i> , and its variety <i>lilacina</i> , <i>I. lacera</i> , <i>I. patouillardii</i> , <i>I. pudica</i> , and many other species of <i>Inocybe</i> .	Mushrooms, urine. <i>Amanita muscaria</i> and related varieties. Discriminate in selecting edible varieties (positive identification).	Avoid eating Clitocybe and <i>Inocybe</i> species. Discriminate in selecting edible varieties (positive identification.)
Ibotenic Acid - Muscimol Poisoning (42, 90)	Ibotenic Acid - Muscimol	Ibotenic acid, muscimol, muscazone, and other compounds. Sedative-hypnotic action. Flycidal effect. Anticholinergic effect. Atropine is contraindicated.	Central nervous system effects. Sedative-hypnotic action. Flycidal effect. Anticholinergic effect. Atropine is contraindicated.	1/2 to 2 hours. Light-headedness, dizziness, uncoordination, drowsiness or sleep, followed by state of excitement, restlessness, confusion, delirium, disturbances of vision (blurred vision), "mind-expanding" visions, muscle spasms, inability to perform certain acts, clouded sensorium, partial amnesia, vomiting may or may not occur.	Mushroom tissue. Extract, chromatography, <i>(panther fungus)</i> , <i>A. cothurnata</i> , <i>A. gemmata</i> , <i>A. smithiana</i> , <i>A. strobiliformis</i> , <i>Tricholoma muscarium</i> .	Mushrooms; ⁹ <i>A. muscaria</i> (fly agaric), <i>A. pantherina</i> (panther fungus), <i>A. cothurnata</i> , <i>A. gemmata</i> , <i>A. smithiana</i> , <i>A. strobiliformis</i> , <i>Tricholoma muscarium</i> .	Avoid eating <i>Amanita muscaria</i> and related varieties. Discriminate in selecting edible varieties (positive identification).

⁸ Suspected etiologic agents: *A. gemmata*, *A. parvovolvata*, *Boletus calopus*, *B. luridus*, *B. pulcherrimus*, *B. satanus (eastwoodiae)*, other spp. of *Boletus*, *C. (Higrophorus) aurantiaca*, *C. nebularis*, *Hebeloma crustuliniforme*, *Mycoena pura*, *Omphalotus (C) olearius (illidens)*, *Russula emetica*.

⁹ Suspected etiologic agents: *A. cokeri*, *Panaeolus campanulatus*, *P. refutans*.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Psilocybin - Psilocin	Psiilocybin, Psiilocin, Hallucino-genic Mushroom Poisoning (42, 90, 284)	Affects central nervous system. Psychotomimetic manifestations. Oral dose 4-8mg (2g dried mushroom room). Psiilocin sensitive to oxidation; psiilocybin activity retained in dried mushroom and extracted into boiling water.	1/2 to 3 hours, usually 30 to 60 minutes. Anxiety, difficulty concentrating and understanding, changes in sensory perception, mood elevated or depressed, laughter, hallucinations. In children, high fever and tonic-clonic type convulsions may develop. Resembles symptoms induced by lysergic acid diethylamide (LSD). Recovery in 5 to 10 hours.	Mushroom tissue. Some used in native magico-religious ceremonies. Usually grows on or near dung.	Mushrooms: 10 <i>Psiilocybe baeocystis</i> , <i>P. caeruleipes</i> , <i>P. caerulipes</i> , <i>P. cyanescens</i> , <i>P. cubensis</i> , <i>P. pelliculosa</i> , <i>P. semianulata</i> , <i>P. strictipes</i> , <i>P. stuntzii</i> , <i>Panaeolus castanifolius</i> , <i>P. (Copelandia) cyanescens</i> , <i>P. fimicola</i> , <i>P. foenisecii</i> , <i>P. sphinctrinus</i> , <i>P. subhalteatus</i> , <i>Conocybe cyanopus</i> , <i>C. smithii</i> , <i>Gymnopilus aeruginosus</i> , <i>G. validipes</i> .	Mushrooms, urine. Extract, chromatography. Identification of mushroom. ⁵	Avoid eating the listed varieties of mushrooms. Boil and discard water.
Coprine Poisoning (Mushroom Alcohol Intolerance) (42,47,90)	Disulfiram-like (antabuse) constituents and alcohol.	Amino acid. Interferes with normal metabolism of alcohol. Chelating properties that bind molybdenum, block acetaldehyde dehydrogenase that arrests ethanol metabolism. Vasoconstrictor effects. Sensitization requires about 3 to 6 hours.	1/2 to 2 hours. Flushing (purple-red face), metallic taste, paresthesia of extremities, palpitation, dyspnea, hyperventilation, tachycardia, feeling of swelling of hands. Lacer, nausea, vomiting, sweating. Attacks may recur if alcohol is consumed even after 48 hours. Duration 30 minutes to a few hours.	Mushroom tissue and alcohol. Several drinks of alcohol before ingestion or one drink within 5 days of ingesting C. atramentarius.	Inky cap mushrooms. <i>Coprinus atramentarius</i> ¹¹ and alcohol. Citocyte claviceps and beer or sake.	Mushrooms. Chromatography. Identification of mushrooms. ⁵	Abstain from alcohol for several days after eating C. atramentarius or avoid eating C. atramentarius.

¹ Suspected etiologic agents: *Amanita citrina*, *A. porphyria*, *Boletus manicus*, *Clitocybe gallinacea*, *Conocybe siligineaoides*, *Gymnopilus purpuratus*, *G. spectabilis*, *Lycoperdon marginatum*, *Naematoloma porporinum*, *Panaeolus campanulatus*, *P. nivalis*, *Phallaceum (solidipes)*, *P. reticulatus*, *P. semiovatus*, *(separatus)*, *Psathyrella sepulchralis*, *Russula nondorbiini*, *Stropharia aezuginosa*, *S. coronilla*, *S. homemannii*, *S. squamosa*.

¹¹ Suspected etiologic agents: *C. micaceus*, *C. fuscescens*, *C. insignis*, some African species of *Coprinus*.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Mushroom Poisoning - Gastro-intestinal Type (42, 45, 47, 90)	Unknown - probably a variety of toxic substances.	Gastrointestinal irritants. There is little if any liver involvement, acute nervous system stimulation, or pronounced psychologic disturbance.	1/4 to 4 hours or longer, usually less than 2 hours. Nausea, vomiting, diarrhea, abdominal cramps and pain. Duration 1 to 2 days.	Mushroom tissue. Some are not poisonous to all people. The same variety may be eaten without ill effects on one occasion and cause illness the next.	Certain mushroom species and genera, including <i>Agaricus albolutescens</i> , <i>A. arvensis</i> , var. <i>Pulustis</i> , <i>A. hondensis</i> , <i>A. placomyces</i> , <i>A. xanthodermus</i> , <i>A. silicola</i> . ¹²	Mushrooms.	Avoid eating the listed varieties. Discriminate in selecting edible varieties (positive identification).
Mushroom Poisonings - Unclassified (40, 80)				<i>Hippolyoma (Naematozoma) fasciculare</i>			Avoid eating this species. Discriminate in selecting edible varieties (positive identification).
				<i>Paxillus involutus</i>			Avoid eating this species. Thorough cooking.
					<i>Clitocybe toxica</i>		Avoid eating this species. Discriminate in selecting edible varieties (positive identification).

¹²Other species that have been implicated are *Amanita brunneescens* and its variety *pallida*, *A. chlorinosma*, *A. flavoconia*, *A. frostiana*, *A. parvivolvata*, *A. rubescens* (raw), *A. spreta*, *A. vaginata* (raw), *Boletus flocosus*, *B. luridus*, *B. pulcherrimus*, (*B. eastwoodiae*), *B. satanas*, *B. sensibilis* (miniate-olivaceous), *Chlorophyllum molybdites*, *Clavaria formosa*, *C. gelatinosa*, *Clitocybe*, (*Rhodophyllus*) *livium* (*sinuatus*), *E. (Nolanca) mammosum*, *E. nidorsum*, *E. pascuum* (*straurospora*), *E. rhodopollum*, *E. salmonicum*, *Entoloma* (*Rhodophyllus*) *livium* (*sinuatus*), *E. (Nolanca) mammosum*, *E. fastibile*, *H. mesophaeum*, *H. sinapizans*, *E. vernum*, *Gomphus (cantharellus) flocosus*, *G. bonarii*, *G. kauffmanii*, *Hobeloma crustuliniforme*, *H. fusibus*, *L. glaucescens*, *Lactarius torninosus*, *L. travigalis*, *L. velleatus*, *L. chrysoneurus*, *L. helvus*, *L. rufus*, *L. representaneus*, *L. uvidus*, *L. naucina* (*Leucoagaricus naucinus*), *Lycoperdon marginatum*, *Lepiota clypeolaria*, *L. cristata*, *L. lutea*, *Leucocoprinus luteus*, *L. morgani*, *L. naucina* (*Leucoagaricus naucinus*), *Lyopholoma (Lyopholoma) fasciculare*, *L. subincarnatum*, *Morchella angusticeps* (elata), *M. crassipes*, *M. deliciosa*, *M. semilibera*, *Naematoloma* (*Abatrellopsis*) *berkeleyi*, *P. (Albatrellus) pessundatum*, *Morchella esculenta*, *M. esculenta*, *M. semilibera*, *M. squarroso*, *Polyporus (Bondarzewia) berkeleyi*, *P. (Lactiporus) sulphureus*, *Ramaria (Clavarria) formosa*, *R. gelatinosa*, *Russula emetica*, *R. foetens*, other *Russula* spp., *Scleroderma aurantium* (*citrinum*), *S. cepa*, *Tricholoma album*, *T. muscarium*, *T. nudum* (*Citocybe nuda*) (raw), *T. pardinum*, *T. pessundatum*, *T. saponaceum*, *T. sejunctum*, *T. sulphureum*, *T. vescinum*, *T. venenata*, *Verpa bohemica*.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
Mushroom Poisonings - Unclassified (continued)		Muscular incoordination		<i>Verpa bohemica</i>			Avoid eating this species. Discriminate in selecting edible varieties (positive identification).
			Tetany and pataesthesia	<i>Scleroderma aurantium</i>			Avoid eating this species. Discriminate in selecting edible varieties (positive identification).
			<30 minutes	<i>Scleroderma cepa</i>			Avoid eating this species. Discriminate in selecting edible varieties (positive identification).
			Stomach pain, weakness, nausea, tingling over entire body, muscular rigidity, sweating, facial pallor.	<i>Stropharia coronilla</i>			Avoid eating this species. Discriminate in selecting edible varieties (positive identification).
Phycomycosis (Mucormycosis, Zygomycosis) (3)	<i>Absidia</i> , <i>Rhizopus</i> , <i>Mortierella</i> , <i>Basidioholobus</i> , <i>Mucor</i> , <i>Cunninghamamella</i> spp.	Common saprophytic fungi. Opportunistic and able to cause infection because of localized or systemic factors that lower tissue resistance.	Few days. Intestinal form: Abdominal pain, diarrhea, bloody stools, mucosal ulcers, thromboses and gangrene of stomach or bowel, hematemesis, peritonitis. More frequently invades other sites: lungs, facial sinuses, brain, skin.	Soil, decaying vegetation, and moldy food. Frequently associated with people who have diabetes mellitus, malnutrition, or other illnesses. Only a few cases of macro-mycosis of digestive system on record. Airborne transmission more likely.			Blood, feces, biopsy or autopsy tissues. Microscopy, isolation, identification.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
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PLANT TOXICANTS AND TOXINS (38, 41, 48, 53-64)

ALKALOIDS							
Jimson Weed or Nightshade Poisoning (41, 55, 57)	Tropane (Belladonna) alkaloids: Atropine, Hyoscyamine, ¹³ Scopolamine (Hyoscine).	Stimulant, mydriatic, parasympatholytic action; blocks motor, secretory, and inhibitory effects of acetylcholine on smooth muscle tissue. Cerebral convulsant.	<1 hour. Abnormal thirst, photophobia, dilated pupils, distorted sight, difficulty speaking; hot, dry, flushed skin; rash, delirium, incoherence, coma, rapid heartbeat, cyanosis, lassitude, nausea, hallucination.	Seeds, roots, leaves, all parts of plants. Grafting tomato plants to Jimson weed stock caused tomatoes to become toxic.	Jimson weed, Jamestown weed, thorn apple (Datura stramonium), D. meteloides, deadly nightshade (<i>Atropa belladonna</i>). Tea made from leaves. Grafted tomatoes.	Urine, stomach contents, plant.	Avoid eating any part of these plants. Become familiar with poisonous plants in area.
Senecio Poisoning (Venocclusive Disease; Bread Poisoning) (57, 60, 64)	Pyrrrolizidine alkaloids.	Inhibits neuromuscular function. Not destroyed on drying or during silage fermentation. Carcinogens and tumorogens. Alkylating agent. Heat stable.	Dyspepsia, ascites, enlarged liver, abdominal pain, nausea, vomiting, headache, anaphy, emaciation, diarrhea. High case fatality rate.	Seeds.	Groundsel (<i>Senecio spp.</i>) Medicinal (gordolobo) tea, bread made from flour contaminated with seeds. <i>Crotalaria</i> nanaburn millet (gondili)	Urine, plant. Extract, chromatography, colorimetry, crystal form.	Avoid eating ground-seed seeds. Detoxify, separate weed fields, separate contaminated grains by sieving.
Hemlock Poisoning (55, 103)	Pyridine alkaloid: Coine and related compounds. Juice is the Greek hemlock potion.	Blocks spinal reflexes by action on spinal cord. Odor of parsnip.	<1 hours. Nervousness, trembling, ataxia, muscular weakness, dilation of pupils, slowed heartbeat, thirst, coldness, nausea, vomiting, convulsions, coma, respiratory failure.	Utripe fruit, plant, root (conium seed). Quail feeding on this plant has been suggested as a cause of quail poisoning.	Urine, plant. Extract, chromatography, colorimetry, crystal form.	Poison of deadly hemlock, poison parsley (Conium maculatum). Fool's parsley (<i>Aethusa cynapium</i>) contains the same alkaloids. Mistletoe taken for parsley.	Avoid eating any part of these plants.

¹³ Black henbane (*Hyoscyamus niger*) also contains hyoscyamine.

¹⁴ History of eating plants involved and identification of plants are important in diagnosis.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD / SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY ¹⁴	CONTROL MEASURES
Nicotine Poisoning (41, 55, 58)	Nicotine Lotelline Anabasine	(See poisonous chemicals, page 66 for detailed description.)	Cardinal flower (<i>Lobelia cardinalis</i>) and tree tobacco (<i>Nicotiana glauca</i>)	Avoid eating tobacco leaves or snuff.			
Epidemic Dropsy (64, 250)	Areogenone oil. Isoquinolippe alkaloid; ¹⁵ Sanguinarine, Berberine, Protopine.	Irritates mucous membranes, depresses smooth muscles. Dilatation of capillaries. Interferes with oxidation of pyruvic acid.	Edema (particularly in low extremes), diarrhea, vomiting, pyrexia, anemia, dyspnea, tachycardia. Myocardial involvement.	Seeds and whole plant. Persons with low protein intake are more susceptible.	Prickly poppy (<i>Argemone mexicana</i>). Contaminated mustard oil (cooking oil), peanut oil, wheat.	Urine, plant. Extract, chromatography, colorimetry, crystal form. Animal feeding tests.	Avoid eating any part of prickly poppy. Purchase cooking oil from reliable source. Increase dietary protein.
Mescal Bean or Laburnum Poisoning (41, 55, 59)	Quinolizidine alkaloid; Cystisine	Similar to nicotine. Stimulation followed by depression of respiratory system	<1 hour. Salivation, nausea, vomiting, retching, diarrhea, delirium, prostration, respiratory paralysis, collapse, convulsions, coma, visual disturbances.	Seed and bean-like capsules containing seeds.	Mescal, coral bean, or Texas mountain laurel (<i>Sophora secundiflora</i>), laburnum, goldenchain (<i>Cytisus Laburnum</i>) scotch bloom. Milk from cows feeding on plants.	Leaves and seed pods. Extract, chromatography, colorimetry, crystal form.	Avoid eating leaves and seeds of mescal and laburnum. Prevent milk cows from grazing in areas containing scotch bloom.
Solanine Poisoning (55, 57, 60, 64)	Solanaceous alkaloid; Solanine ¹⁶	Cholinesterase inhibitor. Exposure to sunlight increases solanine content.	1 to 6 hours. Burning of throat, nausea, stupefaction, convulsions, diarrhea, collapse, delirium, lassitude, muscular weakness.	Urripe, globular berries, leaves, sprouts, eyes, skin.	Bittersweet (Solanum dulcamara), black nightshade (<i>S. nigrum</i>) ^{16, 17}	Urine, plant. Alkaline, extract, chromatography, colorimetry.	Avoid eating these plants. Cook solanine-containing foods thoroughly.

¹⁵Bloodroot (*Sanguinaria canadensis*) also contains Sanguinarine.

¹⁶Other plants containing solanine include Jerusalem cherry (*S. pseudocapsicum*), horse or bull nettle (*S. carolinense*), Silver leaf nightshade (*S. elaeagnifolium*), Sodom apple (*S. sodomae*), three-flower nightshade (*S. triflorum*), Day-blooming Jessamine (*Cestrum diurnum*), Night-blooming Jessamine (*Cestrum nocturnum*).

¹⁷Solanine in minute amounts in potato sprouts.

DISEASE	Etiologic Agent	Nature of Organism/Toxin	Incubation (Latent) Period/ Signs and Symptoms	Source, Reservoir and Epidemiology	Foods Involved	Specimens / Laboratory ¹⁴	Control Measures
Green Hellebore or Death Camas Poisoning (57,60)	Steroidal, veratrum alkaloids; Veratridine, Veratrine.	Medullary excitant. Effects heart muscles and respiration. Hypotensive effect.	Burning sensation of mouth and throat, salivation, vomiting, abdominal pain, retching, visual disturbances, cold and clammy skin, depressed circulation, hallucinations, headache, prostration.	Roots, bulbs, leaves, seeds. Mistaken for onion.	Green hellebore, false hellebore, Indian poke (<i>Veratrum viride</i> , <i>V. japonicum</i> , <i>V. californicum</i>). Death camas (<i>Zygadenus spp.</i>) Flour and soup made from green hellebore.	Plant. Extract, chromatography, colorimetry, crystal form.	Avoid eating these plants.
DeLphinium or Monkshood Poisoning (41,55,57)	Poly cyclic diterpene alkaloids: Ajacine, Ajaconine, Delphinine, Aconitine.	Hypotensive action. Affects nervous system (vagus nerve).	<1 hour. Tingling sensation of mouth, collapse, lassitude, prostration, visual disturbances, slow pulse, labored respiration, stiffness of facial muscles, irregular muscle twitching, depression, nausea, vomiting. Asphyxiation and respiration paralysis is responsible for death which occurs in a few hours.	Roots, seeds, leaves. Toxicity decreases with maturity of plant. Grows in stony ground.	Larkspur, lark's claw, knight's spur (<i>Delphinium ajacis</i> and related species.) Monkshood aconite, friar's cap, wolfsbane (<i>Aconitum napellus</i> and related species.) mistaken for horseradish.	Plant. Extract, chromatography, colorimetry, crystal form.	Avoid eating these plants.
Daffodil Bulb Poisoning (41,55)	Alkaloid: Lycorine	Heat stable.	Vomiting, shivering, diarrhea.	Bulb.	Daffodil (<i>Narcissus pseudo-narcissus</i>). Mistaken for onion.	Urine, plant or bulb.	Avoid eating the bulbs.
Yew Poisoning (41,55,57)	Alkaloid: Taxine	Cardiac depression, gastrointestinal irritant.	<1 hour. Nausea, vomiting, diarrhea, abdominal pain, dizziness, dry throat, muscular weakness, stupor, prostration, circulatory slowing and failure, difficulty breathing, collapse, sudden death.	Seeds, berries, foliage, needles, bark. All parts of plant.	Yew (<i>Taxus baccata</i> , <i>T. canadensis</i> , <i>T. cuspidata</i> , <i>T. brevifolia</i>).	Leaves, berries, urine.	Avoid eating any part of this plant.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
Strychnine and Carolina Jessamine Poisoning (41, 55, 57, 75)	Alkaloid: strychnine and compounds related to strychnine.	Depressant.	<1 hour. Convulsions, clonic phase, spasm, depression, respiratory failure, death within 24 to 48 hours.	Rodenticide bait. Nectar from flowers.	Strychnine tree (Strychnos nux-vomica), Carolina jessamine (Gelsemium sempervirens). Honey.	Urine, plant. Separation, chromatography.	Avoid eating rodent bait and nectar from Carolina jessamine.
Colchicine Poisoning (41)	Alkaloid: Colchicine	Mitotic poison, depression of cell division.	2 to 6 hours. Burning of mouth and throat, strangling, dysphagia, intense thirst, nausea, abdominal pain, violent uncontrollable vomiting, diarrhea, tenesmus, shock, collapse, hematuria, oliguria, respiratory paralysis.	All parts of plant.	Glory lily (Gloriosa superba), Autumn crocus (Colchicum autumnale), leaves in salads, tubers mistaken for yams, seed pods, milk of poisoned livestock.	Urine, plant. Extract, chromatography, colorimetry, crystal form.	Avoid eating any part of these plants.
Peyote Poisoning (55, 199)	Alkaloid: Mescaline from peyote cactus	Hallucinogen, 350-500mg.	Changes in spatial and temporal consciousness. Visual hallucinations, anxiety, hyper reflexia of limbs, static tremors, nausea, vomiting.	Desert plant.	Peyote or mescal buttons (Lophophora williamsii).	Urine.	Avoid eating peyote buttons.
Morning Glory Poisoning (55)	Alkaloids: Lysergamide, Isergotine, Elymoclavine, and others	Hallucinogen.	Seeds	Morning Glory (<i>Ipomoea violacea</i>)	Urine, plant, seeds. Extract chromatography	Avoid eating Morning Glory seeds.	

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
Glycosides							
Cyanide Poisoning (55, 59, 60)	Cyanogenic glycosides: Amygdalin, Pronasin, Pruhaurisin	Liberates hydrogen cyanide; odor of bitter almonds. Soaking in water hydrolyzes the cyanogen in cassava.	<1 hour. Asphyxia, dyspnea, vomiting, excitement, gasping, staggering, fibrillary twitchings, paralysis, stupor, convulsions, coma, collapse, cyanosis, lassitude, prostration. Death within 15 minutes to 1 hour with lethal dose.	Seeds.	Bitter almond (Prunus amygdalus), cassava; choke cherry, cherry, peach, wild black cherry, apricot, plum, cherry laurel pits, apple seeds, Pyrus sylvestris, apricot kernel jan, lima beans, red kidney beans, loquat plum, hydrangea buds and leaves (Hydrangea macrophylla).	Plants, stomach contents, blood, urine.	Avoid eating seeds of plants involved. Cook foods thoroughly. Selective breeding of low-cyanide varieties of lima beans to those yielding less than 20 mg HCN/100 g of seed. Soak and ferment cassava.
Glycogenes							
Golter (59)	Gofitrogens: Thiokazolidine derivatives, Thiocyanates. ¹⁵	Interferes with the uptake of iodine by thyroid gland.	Enlargement of thyroid gland. Variable, depends on intake of goitrogenic substances, iodine, and other goitrogen antagonists.	Root, seed, or leaf. Dietary history.	Rutabaga, white turnip, cabbage, soybean, rape seed, peach, pear, strawberry, spinach, carrot. Milk from cows feeding on these plants.	Eat a diet containing iodine and vitamins A and D. Cook foods thoroughly. Freeze foods.	Avoid eating these plants.
Banberry Poisoning (40)	Protoanemonin	Gastrointestinal irritant, unstable oil.	<1 hour. Burning sensation of mouth and pharynx, Inactivated by drying and cooking.	Berries; root stock, sap.	Banberry, dolls eyes (Actaea spp.), buttercups.	Plants.	Avoid eating these plants.

¹⁴Noniodine halides, cobalt, calcium, copper, ergothioneine, cyanoglycosides, polysulfides, and indolylacetonitrile are possible goitrogens.

DISEASE	EPILOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
Buckeye (Horse Chestnut) Poisoning (55,57)	Unknown. Coumarin Glycosides, Aesculin, Daphnin, and Saponin present.	Anticoagulant.	Blood fails to clot.	Nuts, seeds, sprouts.	Buckeye, horse chestnut (<i>Aesculus</i> <i>giabra</i> and related species). Honey made from flower of California buckeye. Tea from young shoots and leaves.	Leaves and seeds.	Avoid eating the nut.
Oleander Poisoning (41,55,57)	Cardiac Glycosides: Thevetin, Convallarin, Steroidal, Helleborein, Quabain, Digitoxin.	Paralyzes sympathetic nerves. Cardiotoxin stimulates heart muscles similarly to digitalis. Produces gastric distress.	1 to 24 hours. Nausea, dizziness, drowsiness, irregular heartbeat, cyanosis, collapse, coma, increased peristalsis, intestinal spasm, vomiting, diarrhea, tingling and numbing sensation of the mouth, visual disturbances.	Kernel of fruit, sticks, wood, nectar, roots, leaves, fruit, seed.	Oleander (<i>Nerium</i> <i>oleander</i> , <i>N. indicum</i>). ¹⁹ Honey made from flowers. Meat roasted on oleander sticks. Milk from cow that ate foliage.	Leaves and flowers. Separation, colorimetry.	Avoid eating any part of these plants. Avoid using oleander sticks as skewers.
Poisoned, Corn Cockle, Finger Cherry, or Chinaberry Poisoning (55,57)	Saponins: Githagenin, Agrestemic Acid.	Emetic and carthartic action, gastrointestinal irritant, hemolytic.	<1 to 2 hours. Burning sensation in mouth, nausea, vomiting, diarrhea, gastro- intestinal cramps, salivation, lassitude, drowsiness, vertigo, prostration, convulsions, visual disturbances, collapse, blindness, collapse, narcosis, paralysis, coma. Recovery usually in 24 hours.	Fruits, roots, shoots, seed. Particularly leaves and green berries.	Poisoned root, green berries, (<i>Phytolacca</i> <i>americana</i>). ²⁰ Githago, corn cockle (<i>Agrostemma</i> <i>githago</i>), finger cherry, loquat (<i>Rhodomyrtus</i> <i>macrocarpa</i>), flour contami- nated with seeds. Chinaberry (<i>Melia</i> <i>azedarach</i>).	Plants.	Avoid eating any part of these plants. Parboil leaves and discard water.

¹⁹Other plants that contain cardiac glycosides are yellow oleander, be-still (*Thevetia peruviana*), lily-of-the-valley (*Convallaria majalis*), Strophanthus (*Strophanthus kombe*), Christmas rose, black hellebore (*Helleborus niger*), foxglove (*Digitalis purpurea*).

²⁰Wild balsam apple (*Momordica balsamina*), baypod (*Sesbania vesicaria*), senna bean (*S. drummondii*), purple rattlebox (*S. punicea*), English ivy (*Hedera helix*), contain saponin.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY ¹⁴	CONTROL MEASURES
Tung Nut Poisoning (6,55)	Saponin (sapotoxin) and phytotoxin	Gastrointestinal irritant. High protein content. Two toxic fractions, one insoluble and heat labile, and the other soluble and heat stable.	<1 hour. Nausea, abdominal cramps, severe vomiting, diarrhea, prostration, dehydration, shock, cyanosis, respiratory depression, diminished reflexes may occur in severe cases.	Seed. Trees are common in Gulf Coast States.	Tung nut (<i>Aleurites fordii</i>)	Leaves and nut	Avoid eating tung seeds or nuts.
Cycas Poisoning (57,66,170)	Azoxo Glycoside Cycasin	Carcinogenic and hepatotoxic. Multiple ingestions usually required.	6 to 24 hours. Nausea, vomiting, unconsciousness, jaundice (swelling of liver). Death within 20 hours.	Seeds or stems of cycas	Ojiva (gruel made of cycas flour and bean paste) <i>Cycas circinalis</i> , <i>C. revoluta</i> .	Vomitus, seed root or flour, Polarography; Chromotography.	Prepare flour properly, steep in water when making starch.
Red Squill Poisoning (48,63)	Glycosides Scillaren A and B	Cardiotonic and emetic action. Large dose required.	Vomiting, abdominal pain, blurred vision, cardiac irregularity, convulsions	Bulb, rodenticide	Rodent bait	Vomitus	Avoid eating rodent bait
Toxalbumins							
Castor Bean or Jequirity Poisoning (53,55,57,163)	Toxalbumins, hemagglutinins ²¹ (phytotoxins); Ricin in castor bean, Abrin in jequirity.	Ability to agglutinate red blood cells and hemolytic. Antigenic. Gastrointestinal irritant, cathartic.	1 to 3 days. Burning sensation in mouth, nausea, vomiting, abdominal pain, diarrhea, lassitude, incoordinated movements, vascular collapse, kidney damage, convulsions, stupor, dyspnea, visual disturbances.	Castor bean fruit, press cake and foliage to a lesser extent. Jequirity bean, crab eye, rosary pea, or prayer bead (<i>Abrus precatorius</i>). core contains more abrin.	Castor bean (Ricinus communis), jequirity bean, crab eye, rosary pea, or prayer bean (<i>Abrus precatorius</i>).	Plants and seeds.	Avoid eating seeds (beans). Avoid putting beans made of jequirity beans into mouth.
Favism (6,57,59)	Vicine, a nucleoside.	Susceptibility associated with a sex-linked gene. Allergic reaction. Causes hemolysis of erythrocytes.	5 to 24 hours. Headache, dizziness, vomiting, nausea, diarrhea, prostration, icterus, hemoglobinuria, hemolytic (glucose-6-phosphate dehydrogenase deficiency) anemia. Sometimes fatal.	Inhalation of pollen also causes response in allergic people. Susceptibility genetically controlled.	Bean. Inhalation of pollen also causes response in allergic people. Susceptibility genetically controlled.	Blood, urine, beans.	Cook fave beans thoroughly. Avoid eating fava bean if allergic to it.

²¹Other toxalbumins - Crotin from *Croton tiglium* (also contains a resin), curcin from *Jatropha curcas* (physic nut), and robin from *Robinia pseudoacacia* (black locust).

DI SEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS AND PLANTS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
Resins							
Water Hemlock Poisoning (55, 56, 60)	Resin: Cicutoxin.	Unsaturated aliphatic alcohol. Acts on central nervous system. Medullary excitant.	15 to 60 minutes. Salivation, nausea, severe stomach pain, mental excitement and frenzy, vomiting, frothing at mouth, irregular breathing, tremors, violent convulsions, delirium, respiratory paralysis, death.	Roots (other parts also). Roots similar to parsnip and sweet potato.	Water hemlock (<i>Cicuta virosa</i>). ²² Cowbane, beaver poison, wild parsley, wild carrot (<i>C. maculata</i>). Mistaken for root vegetable.	Urine, tissue, plant.	Avoid eating any part of these plants.
Mountain Laurel, Rhododendron, or Azalea Poisoning (41, 55, 57)	Resin: Andromedo-toxin	Emetic.	4 to 6 hours. Salivation, malaise, vomiting, diarrhea, Tingling of skin, muscular weakness, headache, visual difficulties, coma, convulsions.	Shoots, leaves, and twigs.	Mountain laurel (<i>Kalmia latifolia</i>), Ledum (Labrador Tea), Rhododendron, Ericaceae spp. Honey made from flowers.	Leaves and flowers.	Avoid eating nectar or other parts of these plants.
Other Toxicants, Toxins, and Allergens ²³							
Milk Sickness (6, 53, 55, 60, 118)	Tremetol and resin acid.	Higher alcohol. Liver poison.	< 24 hours. Gradual onset. Weakness, nausea, prostration, loss of appetite, abdominal pain, vomiting, muscular tremors, acetone breath, delirium, coma, death. Hypoglycemia, acidosis occur.	Leaves and stems of plants. Causes trembles in cattle.	Milk, butter, possibly meat from cows feeding on snakeroot root, white snakeroot (<i>Eupatorium rugosum</i>). Rayless goldenrod (<i>Aplopappus heterophyllum</i>).	Urine, blood. Liver and kidney at autopsy.	Prevent animals from grazing in areas containing snakeroot plants.
Cocculus Poisoning (61)	Picrotoxin.	Non-nitrogenous substance. Stimulates central nervous system. Convulsant.	< 30 minutes. Burning pain in throat, vomiting, salivation, diarrhea, giddiness, stupor, unconsciousness, convulsions, asphyxia, heart failure, sudden death.	Seeds.	<i>Cocculus indicus</i> , fish berries, levant nut (<i>Anamirta paniculata</i>). Adulterated beverages.	Plants or berries.	Avoid eating berries of coccus.

²²Other *Cicuta* spp. roots are toxic.

²³There are many other toxic plants that are not listed in this text; some are common house and garden plants. See references for additional data.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS AND PLANTS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Ackee Poisoning (Vomiting Sickness) (55)	Hypoglycin A and B (toxic peptide).	Hepatotoxins. Hypoglycemic effects. Soluble in water.	2 to 6 hours. Two forms: 1) Vomiting. Period of remission, 8 to 10 hours, secondary vomiting, epigastric discomfort, convulsions, coma. 2) Convulsions, coma (unconsciousness with restlessness and irritation to deep depression). Acidosis, low blood sugar and altered pH of blood. Mortality 40 to 80%.	Unripe fruit, seeds. A tropical tree. Reported more frequently in Jamaica. More commonly affects undernourished children.	Unripe Ackee (<i>Bilighia sapida</i>).	Blood, liver biopsy. Ackee fruit.	Avoid eating unripe fruit. Cook and discard cooking water. Edible when handled properly. Eat adequate diet.
Lathyrism (59, 64, 95)	Lathyrogens (Amino-propionitrile).	Lathyrogenic. Protoplastic poison.	4 to 8 weeks. Muscular weakness, paralysis of legs, spastic paraplegia, tremors, osteoporosis, skeletal changes. Posture is with feet turned in and toes down.	Pea. Occurs in people with prolonged and monotonous diet of <i>Lathyrus</i> . Associated with conditions of poverty and drought.	White vetch, vetchling, grass pea (<i>Lathyrus sativus</i>); sweet pea (<i>L. odoratus</i>).	Plants.	Steep peas in hot water; discard water. Cook in large amount of water, discard excess. Eat varied diet.
Oxalate Poisoning (55, 57, 59, 75, 175)	Oxalic acid. Soluble oxalates. Illness perhaps due to a glycoside, anthraquinone.	Corrosive. Combines with blood calcium.	2 to 48 hours. Nausea, vomiting, abdominal pain, bloody diarrhea, stupor, swollen abdomen, headache, somnolence.	Rhubarb leaves (<i>Rheum rhaboticum</i>), sorrel grass, beet tops, Indian turnip (Jack-in-the-Pulpit), dieffenbachia (Dumb Cane), dragon root, elephant's ear, skunk cabbage, American ivy (<i>Parthenocissus quinquefolia</i>).	Leaf, blade.	Urine, plants. Extraction, precipitation, microscopic examination of crystals.	Recognition that rhubarb and other leaves of listed plants are poisonous. Avoid eating these leaves.
Manchineel Poisoning (51)	Unknown (perhaps an indole alkaloid).	Caustic.	Fruit, milky sap.	Leaves and fruit.	Manchineel (<i>Hippocratea mancinella</i>).		Avoid eating any part of manchineel.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS AND PLANTS INVOLVED	SPECIMENS/ LABORATORY ¹⁴	CONTROL MEASURES
Mistletoe Poisoning (55,57)	Choline, amines, phoratoxin.	Cholinergic, adrenergic.	Nausea, convulsions, prostration, coma.	Berries.	Mistletoe (<i>Phoradendron flavescens</i>), tea brewed from berries.	Leaves and berries.	Avoid eating mistletoe berries.
Nutmeg Poisoning (59,229)	Myristicin (phenol)	Stimulant, narcotic, echoic, hallucinogenic, gastroenteric irritant. Volatile oil.	1 to 6 hours. In sufficiently large doses: burning abdominal pain, euphoria, delirium, and later stupor. Low blood pressure, shock, acidosis.	Seed.	Nutmeg (<i>Myristica fragrans</i>).	Seeds or powder.	Avoid excessive use of nutmeg.
Leucaena glauca Poisoning (59,64)	Mimosine, an alpha-amino-propionic acid.	Plant able to extract selenium from soil.	Can be less than 48 hours. Loss of hair, localized edema. Feces are red color.	Koa haole (<i>Leucaena glauca</i>). Soup containing plant.	Feces, plant.	Feces, plant.	Avoid eating any part of plant. Cook in iron vessel.
Djenkolic Poisoning (64)	Djenkolic acid, an amino acid.	Anuric; urine contains blood, epithelial cells, white crystals; disagreeable body odor.	Seeds (beans).	Djenkol (<i>Pithecellobium lobatum</i>).	Urine, seeds or plants.	Urine, seeds or plants.	Avoid eating djenkol beans.
Lantana Poisoning (55)	Lantadene A and related triterpenoids.	Few hours. Vomiting, diarrhea, weakness, ataxia, photophobia, lethargy, respiration slow, deep, labored. Pupils initially dilate then become pinpoint. Depressed deep tendon reflexes. Cyanosis, coma.	Berries, particularly the green berry.	Lantana, hen and chicken (Lantana spp.).	Vomitus, berries.	Vomitus, berries.	Avoid eating Lantana berries.

DISEASE	ETIOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS AND PLANTS INVOLVED	SPECIMENS / LABORATORY ¹	CONTROL MEASURES
Plant-irritant poisonings (55)	Generally unknown Ilicin	Irritants	30 minutes to 1 hour. Nausea, abdominal pain, severe vomiting, diarrhea, de-hydration.	Bulb	Hyacinth (<i>Hyacinthus orientalis</i>), Holly (<i>Ilex</i> spp.), Yaupon (<i>Ilex vomitoria</i>), Iris (<i>Iris</i> spp.)	Plant, vomitus. Colorimetry.	Avoid eating portion of plant containing toxic principle.
Carotenemia (178)	High concentrations of carotene.		Yellow-orange discoloration of skin.	Bulb Berries Root stock, "bulb" Berries, leaves (?)	Ligustrum (<i>Ligustrum</i> spp.)	Plant.	Avoid eating excessive amounts of yellow vegetables and fruits.
Red Kidney Bean Poisoning (222,234)	Unknown but contains protease inhibitor, trypsin inhibitor, hemagglutinins, cytotoxins, goitrogen, cyonogenic glycoside.	Hemagglutinating lectin	1-2 hours. Abdominal distension, nausea, vomiting, diarrhea, general weakness, recovery within a day.	Uncooked (or cooked at low temperature) water-soaked kidney beans.	Red kidney beans (<i>Phaseolus vulgaris</i>).	Plant. Extraction, chromatography. Beans. Test for hemagglutinins.	Cook red kidney beans.
Esophageal Cancer (?) (212)	Tannic acid.	Carcinogen causing chronic irritation of mucous membranes of the throat.	Throat cancer.	Epidemiologic evidence.	High tannic-acid sorghum and grain in Bantu beer and porridge; perhaps the betel nut.	Determine tannic acid content.	Avoid a diet containing high tannic-acid foods.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
TOXIC ANIMALS (54, 58, 64-68)							
Ciguatera Poisoning (65)	Ciguatoxin.	Neurotoxin. Toxin appears to pass through the food chain without losing its lethal activity and without harm to the carrier. Thermosable. Anticholinesterase.	3 to 5 hours, up to 24 hours. Sudden onset. Abdominal pain, nausea, vomiting, watery diarrhea, muscular aches. Tingling and numbness of lips, tongue and throat, metallic taste, dryness of mouth, perioral tingling and numbness. Anxiety, malaise, prostration, dizziness, pallor, cyanosis, chills, profuse sweating, muscular and joint pain may occur. Weakness, dilated pupils, blurred vision, temporary blindness, paralysis; deaths have occurred. Symptoms may vary greatly. Recovery usually within 24 hours, but tingling may continue for a week or more.	Several species of fish near shores and reefs. But, not all species toxic all the time. Warm, temperate to tropical regions. Usu- ally shore or reef fish and bottom feeders. Unusually large fish of a species more commonly involved.	Fish. Eleven orders, 57 families, and over 400 species have been incriminated. Oysters and clams. Perhaps any marine fish may be potential transsector of ciguatoxin.	Fish gonads, liver, intestines, roe, and gonads of tropical fish. Avoid eating unusually large reef fish. There is no reliable method of detecting poisonous fish by their appearance. Neither frying, baking, boiling, broiling, steaming, steaming, drying, salting, nor other ordinary cooking method destroys ciguatoxin.	Avoid eating liver, intestines, roe, and gonads of tropical fish. Avoid eating unusually large reef fish. There is no reliable method of detecting poisonous fish by their appearance. Neither frying, baking, boiling, broiling, steaming, steaming, drying, salting, nor other ordinary cooking method destroys ciguatoxin.
Moray Eel Poisoning (65)	Ciguatoxin or related toxin.	As above.	30 minutes to 24 hours. Similar to above.	As above.	Species of the genus <i>Gymnothorax</i> (moray eel).	As above.	Avoiding eating moray eels.
Ichthyotoxin-hemotoxin.	Destroyed when heated to 60 - 65C. Drying does not affect toxicity. Hemolytic.	Systemic form: Diarrhea, bloody stools, nausea, vomiting, frothing at mouth, skin eruptions, cyanosis, weakness, paralysis, respiratory distress.	Fish serum or blood. Under normal circumstances, the flesh is not toxic.	Raw moray, conger, and anguillid eel blood or serum.	Serum, eel. Animal inoculations, skin or conjunctiva testing.	Avoid ingesting eel serum. Cook eels thoroughly. Care in handling to avoid cross-contamination.	

Scombroïd (See Bacterial Diseases, page 5).

²⁴History of eating fish involved and identification of fish are important in diagnosis.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁴	CONTROL MEASURES
Tetraodon or Puffer Fish Poisoning (65)	Tetrodotoxin	Neurotoxin (paralysis of central nervous system and peripheral nerves). Toxin stable to boiling except in alkaline solution. Water-soluble toxin. Mainly attacks nerve endings by blocking movement of all monovalent cations.	10 to 45 minutes to 3 or more hours. Tingling or prickly sensation of fingers and toes, malaise, dizziness, pallor, numbness of lips, tongue, extremities, ataxia, nausea, vomiting, diarrhea, epigastric pain, dryness of skin, subcutaneous hemorrhage and desquamation, eyes fixed, reflex lost, respiratory distress, muscular twitching, tremor, incoordination, paroxysms, intense cyanosis. Case fatality rate near 60%.	Ovaries, roe, liver, intestines, and skin are most toxic, but flesh may be toxic. Toxicity is highest during spawning period. Important cause of fish poisoning in Japan. A species may be toxic in one location but not in another.	About 90 toxic species of puffer fish (fugu, blowfish, globefish, porcupine fish, molas, burrfish, balloonfish, toadfish).	Gonads, liver, muscle skin, muscle of fish.	Avoid eating puffer fish. If eaten, wash fish thoroughly when caught. Remove skin, viscera, and gonads. The sale of puffer fish in Japan is strictly regulated. Puffer fish cooks and restaurants are licensed.
Clupeoid Poisoning (Clupeotoxism) (65)	Clupeotoxin	Toxin stable to cooking, salting, and drying.	Few minutes.	Viscera most toxic. Fish are plankton (dinoflagellate) feeders. Apparently the fish become toxic at sporadic intervals. Spotty distribution in tropical, insular areas. Blue-green algae may be the source of the toxin.	Herring, sprat, sardine, tarpon, anchovy, bone-fish, and herring-like fish of the Pacific and Caribbean.	Fish.	Avoid eating clupeiform fish from in-shore, tropical areas in the summer. Prohibit sale of suspect fish. Ordinary cooking procedures do not alter the virulence of the toxin.
File Fish Poisoning (164, 246)	Aluterin	Water soluble.	Vomiting, diarrhea, joint ache.	Believed to originate from zonantharian <i>Palythoa tuberculosa</i> which grows on coral reefs.	File fish (Aluteridae) Feeding to pigs or mice.	Fish.	Avoid eating file fish.

DISEASE,	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁴	CONTROL MEASURES
Elasmobranch and Chondrichthyes Poisoning (65)	Unknown. Ciguatoxin?	May be similar to that of ciguatera poisoning. Stable to heat, water soluble. Toxin in flesh may be deactivated by drying.	Within 30 minutes. Nausea, vomiting, abdominal pain, diarrhea, oily stools, pallor, headache, prostration, rapid weak pulse, burning and tingling of lips, tongue, throat, anorexia. Later, visual disturbances, pain, sensation of heaviness of limbs, chest pain, generalized itching, delirium, coma, and death may occur.	Liver, gonads, and sometimes flesh of fish.	Sharks, dogfish, rays, skates. Particularly tropical sharks.	Liver, gonads, oviducts of Elasmobranch.	Avoid eating liver, viscera, and flesh of tropical sharks, skates, and rays.
Chimaeroid Poisoning (65)	Unknown.		6 to 24 hours. Stupefying effect or mental depression.	Viscera (oviducts, liver, roe) most toxic, but flesh also toxic.	Chimaeras; ratfish, elephantfish.	Fish.	Avoid eating ratfish, elephantfish, or other chimaeras.
Cyclostome Poisoning (65)	Unknown (biogenic amine)	Anticoagulant (?), stable to heat (?), and gastric juices.	Few hours. Nausea, vomiting, diarrhea, tenesmus, abdominal pain, weakness for several days.	Skin, slime, mucus, and flesh.	Lampreys and hagfish. Fish-like vertebrates (eel-like form).	Buccal gland secretion. Blood coagulation and hemolysis.	Deslime fish. Soak in concentrated brine for several hours (?).
Gempylid Poisoning (Gempylo-toxism) (65)	Gempylid oil (Ruvettus oil).	Purgative oil containing cetyl alcohol.	Few hours, about 3. Diarrhea, no pain.	Flesh.	Snake mackerels, castor oil fish.	Fish.	Avoid eating snake mackerel or be aware of laxative effect.

²⁴Trunkfishes (*Ostraciontidae*) have been reported to cause unsteadiness of gait, and Grammatid fishes cause unpleasant bitter taste and slight stinging sensation when they contact the tongue.

DISEASE	ETOLOGIC AGENT	NATURE OF ORGANISM/TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁴	CONTROL MEASURES
Hallucinogenic Fish Poisoning (65)	Ichthyosilyeino-toxin	Heat-stable toxin.	10 minutes to 2 hours. Dizziness, loss of equilibrium, ataxia, hallucination, nightmares, mental depression, sensation of tight constriction around chest. Symptoms persist for 24 hours or more.	Head or flesh. The poison is more concentrated in the head. Reef fish. Occurs in tropical Pacific and Indian Ocean regions.	Similar to those causing ciguatera poisoning. Such as mullet, goatfish, rudderfish, surgeonfish, rabbit fish, grouper.	Fish.	Avoid eating tropical reef fish that have been associated with this illness.
Fish Liver Poisoning (65)	Ichthyohepatotoxin.	Hypervitaminosis A believed to be important. Heat-stable toxin.	30 minutes to 12 hours. Nausea, vomiting, fever, headache, mild diarrhea, rash, loss of hair, dermatitis, large areas of skin peel away, bleeding from lips, joint pain.	Liver. Most outbreaks reported in Japan.	Sawara (Japanese mackerel), ioshingai (sea bass, sandfish, porgy).	Urine, fish liver.	Avoid eating liver of these fish.
Fish Roe Poisoning (65,146)	Ichthyotoxin.	Some toxins are destroyed by heat, others are not. Some are lipoprotein.	1 to 6 hours. Bitter taste, dryness of mouth, intense thirst, headache, fever, vertigo, nausea, vomiting, abdominal cramps, diarrhea, dizziness, cold sweats, chills, cyanosis. Paralysis, convulsions, and death in severe cases.	Roe and ovaries. Reported in Europe, Asia, and North America.	Carp, barbel, pike, sturgeons, gar, catfish, tench, bream, minnows, salmon, whitefish, trout, blenny, cabezon, and other freshwater and saltwater fish.	Roe, ovaries. Extraction, mouse injection.	Avoid eating roe of any fish during reproductive season. Feed to animals.
Haff or Yuksoe Disease (252)	Unknown. Thiamin inactivative factor (?) Blue-green algae (?) Mercury (?)		<24 hours. Sudden, acute pains in legs, arms, and back muscles. Slight touch or movement causes cry of pain; vomiting. Urine is brownish-black color.	Flesh. Occurs in lakes following luxuriant bloom of blue-green algae.	Perch, bream, roach, turbot, lake trout.	Algae, water, fish.	Avoid eating fish from lakes having unusually large algae growth or industrial mercury pollution.
Minamata Disease				(See Alkyl-Mercury Poisoning, page 73.)			

DISEASE	ETOLOGIC AGENT	NATURE OF TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁶	CONTROL MEASURES
Paralytic Shellfish Poisoning (Dinoflagellate Poisoning) (65)	Saxitoxin or related compounds.	Neurotoxin blocks neuromuscular junction. Alkaloid. Relatively heat stable. During red tides, cell counts of plankton blooms may reach 20 to 40 million per ml. Produces neuromuscular weakness without hypotension and lacks emetic and hypothermic action of tetrodotoxin. 80 µg of purified toxin per 100 g of tissue may be lethal.	< 1 hour. Tingling or burning and numbness around lips, fingertip; ataxia, giddiness, staggering, drooping, dryness, gripping in throat, incoherent speech, aphasia, rash, fever, respiratory paralysis (usually within 12 hours). Patients often report feeling of lightness, as floating on air. Ingestion of purified toxin per 100 g of tissue may be lethal.	Shellfish	Mussels feed on plankton - dinoflagellates (<i>Gonyaulax catenella</i> , <i>G. tamarensis</i> , <i>Pyrodinium poucheti</i>). Toxic materials are stored in various parts of the body of shellfish. Digestive organs, liver, gills, and siphons contain the greatest concentration of poison during warmer months.	Mussels, cockles, clams, soft-shell clams, butter clams, scallops, shellfish broth. Over 38 species reported to transsect dinoflagellate poison. Bivalve mussels are the most common vehicles.	Restrict mussel gathering from toxic areas (red tides). Post warning signs. Avoid eating mussels from unknown sources. Cooking may reduce poison content, but most methods of cooking (steaming, baking, boiling, and frying) do not remove the danger. Sampling and toxin testing of mussel extracts. Prohibit marketing of shellfish that exceed an average of 400 mouse units/ 100 g and none that exceed 2000 mouse units.
Neurotoxic Shellfish Poisoning (65)	Toxin of <i>Gymnodinium breve</i> .	Heat stable. Stimulates post-ganglionic nerve fibers	< 3 hours. Paresthesia, hot and cold temperature sensations, nausea, vomiting, diarrhea.	Shellfish	Mussels or clams, sea water.	Toxicological tests. Shuck, drain, grind, extract, clarify, mouse inoculation. Identify algae.	Monitor coastal waters for increased concentration of dinoflagellates.
Oyster Poisoning (Asari or Venerupin Poisoning) (65)	Venerupin (asaritoxin).	Stable to heat (boiled for 1 hour and still toxic). Organotropic, affecting mainly the liver.	6 hours to 7 days, usually 24 to 48 hours. Anorexia, abdominal pain, nausea, vomiting, constipation, headache, malaise, nervousness, halitosis, bleeding of mucous membranes of nose, mouth, and gums, delirium. No paralysis. High case fatality rate (33%). Death 24 to 48 hours after onset.	Oysters and short-necked clams (asari), <i>Ostrea (Crassostrea) gigas</i> , <i>Bosinea japonica</i> , <i>Venerupis (Tapes) semidecussata</i> .	Oysters and short-necked clams (asari), <i>Ostrea (Crassostrea) gigas</i> , <i>Bosinea japonica</i> , <i>Venerupis (Tapes) semidecussata</i> .	Control shellfish harvesting. Feed to test animals before eating.	Extract, chromatography, animal inoculations.

²⁶History of eating shellfish is important in diagnosis.

DISEASE	ETOLOGIC AGENT	NATURE OF TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ^{2,6}	CONTROL MEASURES
Shellfish Gastro-enteritis (291)	Fat soluble toxin. (Plankton associated?) (Vibrio para-haemolyticus?) (Norwalk agent?)	30 minutes to 12 hours. Diarrhea, nausea, vomiting, abdominal pain.	Reported in Japan.	Mussels (Mytilus edulis), scallops, Patiropecten yessoensis? Chlamys, nipponeis, anozara	Mussels, scallops. Mouse assay, chromatography (Test for pathogenic bacteria and viruses).	Mussels, scallops.	Control shellfish harvesting.
Callistin Shellfish Poisoning (65)	Choline or histamine.	Allergic-like, heat stable.	Immediately on eating, up to 1 hour. Itching, flushing of face, urticaria, sensation of chest congestion, abdominal pain, nausea, vomiting, dyspnea, cough, asthmatic manifestations, hoarseness, sensation of constriction, paralysis or numbness of throat, mouth, and tongue, thirst, hypersalivation, sweating, chills, fever. Recovery in 1 to 2 days.	Ovary. Spawning season. Outbreaks restricted to Japan.	Callistin shellfish, Callista brevispinonata.	Skin testing (Bacteriological tests.)	Avoid eating callistin shellfish during spawning season.
Abalone Poisoning (65)	Abalone viscera poison.	A photodynamic principle. Causes photosensitization, stable to boiling, freezing, and salting.	Depends on exposure to sunlight. Sudden onset. Burning and stinging sensation over entire body, prickling sensation, itching, erythema, edema, skin ulceration on parts of body exposed to sunlight.	Viscera (liver and digestive gland). <i>Haliotis discus</i> , <i>H. sieboldi</i> .	Japanese abalone. Animal feeding tests. Exposing abalone to sunlight.	Abalone. Clams.	Avoid eating visceras of abalone.
Tridacna Clam Poisoning (65)	Ciguatera(?)	Gastrointestinal vasoconstrictor neurotoxicological disturbances.	Resembles ciguatera poisoning.	Viscera.	Clams.	Check with local inhabitants about history of clam-associated poisoning. If it occurs avoid eating clams.	

DISEASE,	ETOLOGIC AGENT	NATURE OF TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ ²⁷ LABORATORY	CONTROL MEASURES
Whelk Poisoning (65, 246)	Tetramine.	Curate-like effects, tetramine (histamine-like), heat stable. Autonomic ganglionic blocking agent.	1 to 4 hours. Intense headache, dizziness, blurred vision, diplopia, tingling, twitching, weakness of extremities, nausea, vomiting.	Salivary Glands. Reported in Japan but eaten without harm in Europe.	Japanese species of Whelk, Neptunea arctica, N. intersculpta, Babyonia japonica.	Whelk.	Remove salivary glands of whelk before eating. Recognition of poisonous species. Avoid eating these species.
Other Marine Animals							
Cephalopod (65)	Unknown. (May possibly be bacterial - <i>Vibrio parahaemolyticus</i> ?)	Apparently heat-stable toxin.	10 to 20 hours. Nausea, vomiting, diarrhea, abdominal pain, low fever, headache, chills, weakness, paralysis, convulsions. Duration 48 hours.	Unknown.	Squid, octopus (cuttlefish?).	Flesh.	Toxicological, bacteriological tests, animal feeding.
Sea Urchin Poisoning (65)	Unknown.	Apparently formed during reproductive season.	Abdominal pain, nausea, vomiting, diarrhea, migraine-like attacks.	Gonads.	Sea urchins, paracentrotus lividus, Tripeutes ventricosus, Centrechinus antillarum.	Gonads, sea urchins. Extract, animal inoculation. (Bacteriological Tests)	Avoid eating sea urchins during reproductive season.
Sea Anemone Poisoning (65)	Unknown	High molecular weight protein (?)	Few minutes. Gastritis, nausea, vomiting, abdominal pain, cyanosis, prostration, stupor, pulmonary edema, shock.	May be nematocyst apparatus or tissues of tentacles.	Raw sea anemones. Radianthus paumotensis, Rhodactis howesi, Physobrachia douglasi.	Sea anemone.	Cook sea anemones. Avoid eating raw sea anemones.
Sea Cucumber Poisoning (65)	Holothurin.	Steroid glycoside. Soluble in water.	< 1 hour. Little known, but may be fatal.	Poison concentrated in the organs of Cuvier.	Few sea cucumber spp.	Organs of Cuvier of sea cucumbers.	Check with local inhabitants about history of sea cucumbers being toxic. If so feed to animals before eating.

²⁷ History of eating shellfish or marine animals and identification of species are important in diagnosis.

DISEASE	ETOLOGIC AGENT	NATURE OF TOXIN	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁸	CONTROL MEASURES
Horseshoe Crab Poisoning (Miami Poisoning) (65)	Unknown.	Saxitoxin?	Within 30 minutes. Dizziness, headache, nausea, slow pulse, subnormal temperature, vomiting, abdominal cramps, diarrhea, cardiac palpitation, lip numbness, paresis of lower extremities, weakness, aphonia, hot mouth and throat, generalized muscular paralysis, trismus, hypersalivation, drowsiness, coma.	Unlaid green eggs, flesh, or viscera during reproductive season.	Asiatic horseshoe crab, other crabs and lobsters(?)	Crab. Extraction, mouse injection.	Avoid eating horseshoe crab during reproductive season. Check with local inhabitants before eating.
Cocnut Crab Poisoning (65)	Unknown.	Unknown.	Violent gastrointestinal upset, headache, chills, joint aches, exhaustion, muscular weakness.	Coconut crab. Probably from feeding on toxic plants. Generally not toxic.	Coconut crab (Birgus latro).	Crab. Animal feeding.	Check with local inhabitants before eating.
Sea Turtle Poisoning (Cheloniotoxication) (65)	Chelonitoxin.		Pew hours to several days (usually over 24 hours). Nausea, vomiting, upper abdominal pain, diarrhea, weakness, vertigo, facial tachycardia, pallor, sweating, coldness of extremities; dry, burning, sore lips, tongue, and throat; foul breath, difficulty in swallowing, white coating on tongue, may become covered with pin-sized, reddened, pustular papules; tightness of chest, hypersalivation, desquamation, headache, somnolence, coma, death. High case fatality rate (28%).	Greatest concentration in liver, but also in flesh, fat, viscera, and blood. Sporadic. Perhaps poison is derived from toxic marine algae. Most outbreaks from Indo-Pacific region.	Green sea, hawkbill, and leatherback turtles.	Turtle meat. Extract, animal feeding.	Eat tropical turtles with caution. Avoid the liver. Feed suspect turtle meat to animals 24 hours before attempting to

²⁸History of eating marine animals and identification of species are important in diagnosis.

DISEASE	ETIOLOGIC AGENT	NATURE OF TOXIN OR POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY ²⁸	CONTROL MEASURES
Asiatic Porpoise Poisoning (65)	Unknown.		Abdominal pain and distention, swelling and numbness of tongue, loss of vision, cyanosis, numbness of various areas of skin, hypersalivation, greenish tinge of saliva, muscular paralysis. Death is common.	Liver, eyes, blood, viscera, and flesh.	Asiatic porpoise, <i>Neophocaena phocaenoides</i> .	Avoid eating Asiatic porpoise.	
Sei Whale Poisoning (65)	Unknown.	Histamine-like substance.	Within 24 hours. Severe occipital headache, neck pain, flushing and swelling of face, nausea, vomiting, abdominal pain, diarrhea, fever, chills, photophobia, epiphora, erratic blood pressure, desquamation of skin of face and neck.	Liver.	Liver of Sei whale (<i>Balaenoptera borealis</i>). (White whales are also reported as being poisonous).	Urine, liver.	Avoid eating liver of Sei whale.
Hyper-Vitaminosis A (65)	Excessive Vitamin A	Cooking does not destroy toxic principle.	1 to 6 hours. Intense throbbing or dull frontal headache, nausea, vomiting, diarrhea, abdominal pain, dizziness, drowsiness, irritability, weakness, collapse, light sensitivity, convulsions, insomnia, desquamation of skin.	Liver and kidney. Occurs in Arctic regions. Toxicity varies with individual animal.	Sled dogs, Arctic foxes, bearded seals, sea lions, bull rope walrus, polar bears.	Liver, kidney.	Avoid eating liver of animals from cold regions.

Other crab poisonings produce symptoms similar to either ciguatera or tetraodon.²⁹

Non-Marine Animals

Hyper-Vitaminosis A (65)	Cooking does not destroy toxic principle.	1 to 6 hours. Intense throbbing or dull frontal headache, nausea, vomiting, diarrhea, abdominal pain, dizziness, drowsiness, irritability, weakness, collapse, light sensitivity, convulsions, insomnia, desquamation of skin.	Liver and kidney. Occurs in Arctic regions. Toxicity varies with individual animal.	Sled dogs, Arctic foxes, bearded seals, sea lions, bull rope walrus, polar bears.	Liver, kidney.	Avoid eating liver of animals from cold regions.
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²⁹Xanthid crabs: *Zorimus aeneus*, *Platypodia granulosa*, *Atergatis floridus*, *Eriphia* spp.; Angatea spp., *Demania toxica*, *Carpilius maculatus* (H.).

DISEASE	ETOLOGIC AGENT	NATURE OF TOXIN	INCUBATION (LATENT) PERIOD / SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY TESTS	CONTROL MEASURES
Toxic Quail Poisoning (Coturnism) (59, 224, 300)	Eliciting factor, conine alkaloid(?)	Individual possesses a sensitivity possibly caused by enzymatic abnormality. Quail fed hemlock seeds are toxic to dogs. Toxic metabolites accumulated by quail during stress of migration may be responsible.	1-1/2 to 3 hours for active people; 7 to 9 hours for people at rest. Nausea, vomiting, chills, sharp muscular pain, myoglobinuria (red urine), oliguria, anuria, atotenia, partial slow-spreading paralysis.	Exertion and fatigue are predisposing factors that shorten incubation period and aggravate the disease. European and African regions.	Quail.	Quail, urine. Animal feeding.	Avoid eating Middle Eastern Quail when excessively fatigued.

DISEASE	ETOIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
POISONOUS CHEMICALS (53,69-79)							
Metallic Containers							
Zinc Poisoning (70,107,162)	Soluble zinc salts.	Astringent, corrosive, and emetic.	10 minutes to 3 hours. Pain in mouth, throat and abdomen, nausea, vomiting, dizziness, collapse, shock, glottic edema, albuminuria, nemaaturia.	Zinc-coated (galvanized) containers (pots, cans, tubs). Acids convert zinc into soluble zinc salts. ¹	Lemonade, cooked apples, mashed potatoes, spinach, chicken, tomatoes, and fruit punch.	Food container, vomitus, stomach contents, urine, blood, feces.	Avoid using galvanized containers to store or to cook high-acid food or beverage.
Cadmium Poisoning (6,70,75,162)	Inorganic salts of cadmium. (See page for information on chronic cadmium poisoning.)	Gastrointestinal irritant. Kidney and liver damage.	15 to 30 minutes. Nausea, vomiting, cramps, diarrhea, retching, shock, headache, vertigo, sensory disturbances in hands and arms, convulsions, and prostration reported in severe cases.	Cadmium-(rust-proof) plated utensils (ice cube trays, pitchers), red, orange, or yellow glazed pottery or steel. Foods left in cadmium containers for as little as 1-1/2 hours have caused poisonings.	Lemonade, punch, fruit gelatin dessert, pop-sicles.	Food, container, vomitus, stomach contents, urine, blood.	Avoid using cadmium-plated containers to store or to cook high-acid food or beverages.
Antimony Poisoning (6,70,75,162)	Antimony salts.	Irritating to mucous membranes. 18 ppm can cause symptoms.	15 minutes to 1 hour. Bitter taste, nausea, vomiting, abdominal pain, diarrhea, muscular pain, irregular respiration, lower temperature, collapse. Usually mild and short in duration.	Glaze of cheap, gray enameled cooking utensils containing antimony oxide. Opacifier remains dispersed in the vitreous coating. Readily attacked by acids.	Lemonade, punch, fruit gelatin dessert, sauerkraut, pop-sicles.	Food, container, vomitus, stomach contents, urine, blood, feces.	Avoid using utensils containing antimony to store or to cook high-acid food or beverages.
Copper Poisoning (70,75,122,162)	Copper salts.	Emetic, irritant, and astringent.	Few minutes to 8 hours. Metallic taste, nausea, vomiting, diarrhea, abdominal pain, hematemesis, hematuria, convulsions. Vomitus is often blue-green color.	Copper pipes, copper containers, copper cake decorators.	Carbonated beverages, high-acid food. Cakes decorated with copper bands.	Food container, vomitus, stomach contents, urine, blood.	Avoid using copper pipes and containers in contact with high-acid food or beverages. Prohibit use of copper cake decorators.

DISEASE, ETIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Lead Poisoning (Plumbism) (75,158,162)	Lead and soluble lead salts.	Local irritant to alimentary tract in acute poisonings. Organ failure in chronic poisonings.	30 minutes and longer. Metallic taste, burn- ing of pharynx, abdominal pain, vomiting (milky), diarrhea (stools may be bloody or black) or constipation, (formerly used to stop fermentation of wine). Inhalation of fumes more common mode of transmission	Lead containers, lead water pipes, auto radiators used as condensers, putty, pesticides, and lead glazes for pottery. Sugar of lead (formerly used to stop fermentation of wine). Inhalation of fumes more common mode of transmission	High-acid fruits, wine, maple syrup, beer, cider, vinegar, sardines, ice, moonshine whiskey, curry powder.	Food contain- er, vomitus, stomach con- tents, urine, blood, stools, hair.
Tin Poisoning (86,282)	Tin	<1/2 to 2 hours, or longer. Bloating, nausea, abdominal cramps, vomiting, diarrhea, headache.	Tin containers, mostly re-tinned milk containers. Only a few re- ported outbreaks. Nitrates present cause excessive corrosion of tin lining of cans.	High-acid food: cherries, punch, herring, tomato juice.	Food, con- tainer, feces, vomitus, stomach con- tents, urine, blood. Digest sample, gravimetric precipitation or volumetric titration.	Avoid using un- coated tin con- tainers to store high-acid food. Avoid excessive nitratification of crops having high-acid fruits during production.
Iron Poisoning (Bantu siderosis) (3,162)	Iron salts.	Years. Skin (bronzing) pigmentation, liver disease, diabetes, cardiac failure.	Excessive dietary iron intake from iron pots. More frequent in males.	Beer, wine.	Urine, blood, liver biopsy. Photometry.	Avoid excessive in- take of iron. Do not ferment and store beer or wine in iron containers.
Monosodium Glutamate Poisoning (Chinese Restaurant Syndrome) (247)	Flavor intensifier.	Intentional Additives	Monosodium glut- amate. One teaspoon- ful of MSG can cause symptoms in susceptible people if stomach is empty. Women are more susceptible.	Chinese restaur- ant food (soups, chow mein, egg, roll, lobster, boiled rice. sauce).	Foods. Liquify, filter, potentiometry.	Avoid excessive use of MSG. Restrict use of MSG in baby food. Ingestion of food before inges- tion of MSG delays absorption of MSG.

DISEASE	ETIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Nitrite Poisoning (75,141)	Nitrates and nitrates.	Interferes with oxygen-carrying capacity of red blood cells. May form nitrosamines which are carcinogenic in vivo.	1 to 2 hours. Nausea, vomiting, cyanosis (blueness of lips and adjacent areas, fingers near nails), rapid breathing, fall in blood pressure, headache, dizziness, dyspnea, trembling, convulsions may occur, weakness, loss of consciousness, methemoglobinemia, chocolate brown discoloration of blood.	Soil, fertilizer, color developer for processed meat. Also transmitted by rural well water supplies. Lack of acidity in the Spinach and other plants permits nitrate-reducing organisms to grow and convert nitrates to nitrites.	Processed meat and fish, beef-tea. Nitrates mistaken and used for salt. Milk formulas.	Blood, suspect food.	Restrict use to 50 ppm in processed meat. Avoid excessive fertilization. Refrigerate fresh and cooked leafy vegetables.
Niacin Poisoning (233)	Niacin (nicotinic acid), sodium nicotinate.	Causes sudden cutaneous vaso-dilation.	Few minutes to 1 hr. Intense flushing of skin, warm feeling, itching, abdominal discomfort, puffing of face and knees.	Color preservative, vitamin additive.	Meat, cornmeal.	Suspect food. Neutralize, precipitate, colorimetry.	Restrict use as meat preservative. Control amounts of additives and thorough mixing.
Triorthocresyl phosphate Poisoning (256,262)	Triorthocresyl phosphate, triaryl phosphate.	Neurotoxic - paralysis of motor nerve trunks.	5 days to 3 weeks (10 days). Leg pain and tenderness, motor weakness, ungainly high-stepping gait, loss of voluntary movements below knees, residual paralysis - food and wrist drop. May be preceded by nausea, vomiting, abdominal pain or diarrhea. Recovery is slow and incomplete.	Lubricating oil, certain plastic (non-food use) containers, refrigerant for machine guns, hydraulic fluid.	Cooking oil substitute, fluid extract of ginger (Jamaica ginger), apio (parsley extract), contaminated flour.	Suspect foods. Autopsy: (myelin sheath and spinal cord lesions). Biopsy: (gastrocnemius muscle).	Avoid use in foods. Store foods away from poisonous substances.
Phenolphthalein Poisoning (53,159)	Cathartic.		1 to 2 hours. Vomiting, diarrhea.	Coloring agent, pH indicator.	Candy brooms, cake.	Vomitus, food. Color reaction in alkaline water.	

³⁰Bacteria that reduce nitrates to nitrites include: Enterobacteriaceae, staphylococci, pseudomonads, *B. subtilis*, and *C. perfringens*.

DISEASE	ETOIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Arsenic Poisoning (Acute) (70, 75)	Arsenic (trivalent or pentavalent)	Systemic action on capillaries, corrosive, chelates with dithiols. Protoplasmic and vascular poison.	10 minutes to several days. Burning of mouth or throat, metallic taste, vomiting, diarrhea (watery and bloody), borborygmi, painful tenesmus, hematuria, dehydration, jaundice, oliguria, collapse, shock. Headache, vertigo, muscle spasms, stupor, delirium may occur.	Pesticide sprays contaminated food. Week killers.	Fruit and vegetables, urine, blood, hair, nails, feces.	Gastric washings, urine, blood, hair, nails, feces. Digest, isolate arsenic, distill, colorimetry.	Apply only to seedlings. Label and store in an area separate from food.
Arsenic Poisoning (Chronic) (Peripheral neuritis) (70)	Arsenic trioxide	Protoplastic poison. Chromosomal damage.	Several days or weeks. Loss of weight, loss of appetite, nausea, diarrhea alternating with constipation, pigmentation and eruption of skin, hair loss, peripheral neuritis.	Sugar formerly used for brewing beer.	Grain, wine, beer, sugar	Urine, feces. Digest, isolate arsenic, distill, colorimetry.	Avoid eating arsenic-contaminated foods.
Brewers' Cardiomyopathy (162, 202, 211)	Cobalt acetate.	Cutaneous basodilator.	2 months to several months. Sudden onset. Dyspnea, weakness, fatigue, edema, neck vein distention, tachycardia, gallop rhythm, tachypnea, orthopnea, chest pain, cough, congestive cardiac failure.	Improver for head of beer.	Beer.	Blood, urine, feces, hair. Heart muscle at autopsy.	Prohibit use of cobalt compounds in beer, eat adequate protein diet. Avoid continued, excessive consumption of beer.
Potassium Bromate Poisoning (227)	Potassium bromate.	Caustic and nephrotoxic. Slowly excreted.	1/2 to 2-1/2 hours. Nausea, vomiting, abdominal cramps, diarrhea, collapse, convulsions, anuria. Methemoglobin formation and hemolysis may occur.	Bread improver, permanent wave kits.	Bread, cake or sugar (contaminated with chemical).	Blood, urine. Colorimetry or titration. Urinalysis.	Avoid keeping potassium bromate in food storage areas.

DISEASE	ETIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	LABORATORY SPECIMENS/	CONTROL MEASURES
Margarine Disease (Plants Disease, Blaschenkrankheit) (130)	Emulsifier "MEIG"	Allergen.	1 hour to several days. Acute onset. Red macules on face, neck, and hands, spreading to other areas within 2 to 24 hours, especially upper extremities. Occasionally a purple swelling of face, hands, and feet - hemorrhage in center of lesions. Fever, headache, difficulty swallowing; edematous cheeks, lips, gums; blood blisters; lesions around mouth.	Chemical emulsifier keeps fat from spattering during frying.	Margarine.	Blood.	Withdraw product from markets. Prevent use in food products.
Methyl paraben Poisoning (245)	Methyl Paraben.	Preservative and antiseptic.	Few seconds to 2 hours. Burning sensation of mouth, tongue, and lips; numbness, mouth sores, headache, nausea, diarrhea.	Mold retardant used in food, drugs, and cosmetics. Antibacterial agent for mustitis.	Cake icing.	Vomitus. Uv. absorbance; Paper or gas chromatography.	Avoid excessive use of mold retardants.
Diphenyl-hydantoin Intoxication (116)	Diphenyl-hydantoin.		30 to 90 minutes. Fatigue, nausea, changes in perception and coordination, dizziness, headache, diplopia, dry mouth.	Epilepsy drug.	Coffee.	Urine.	
Incidental and Accidental Food Additives							
Organic Phosphorus Poisons (75,166,167, 182,215)	Organic alkyl and/or aryl phosphate esters. Parathion Tetracyethyl Pyrophosphate (TEPP), Carbonophenthion (Fritthion), Disulfoton, Malathion Ronnel ¹	Inhibit cholinesterase and allow accumulation of acetylcholine. Among most toxic chemicals known.	Few minutes to 8 hours. Nausea, vomiting, abdominal cramps, diarrhea, excessive salivation, headache, giddiness, nervousness, blurred vision, weakness, chest pain, tearing, respiratory tract secretions, cyanosis, papilledema, confusion, uncontrollable muscle twitching, convulsions, coma, loss of reflexes and sphincter control.	Insecticides.	Parathion: wheat, barley flour, bread, pastry, cereal, sugar. Carbonophenthion: tortillas. Diazinon: Melted for wine, doughnut mix. Any food accidentally contaminated with these insecticides.	Blood, urine, fat biopsy.	Avoid spraying just before harvest. Label and store insecticides in an area separate from food. Wash or blanch food. Patients respond to atropine sulfate.

¹Methylparathion, Tetram, Sardin, Tabun, Paraoxon, Thane, HEP, Sytox, EPA, DDT, Phorate, Phosdrin, Chlorthion, Deptor, Abate, Coumaphos, Dejay, Guthion, Fester, Octamethyl Pyrophosphoramide (OMP), Demeton, Mevinphos, Disulfoton, Azinphosmethyl, Chlorfenphos, Dichlorvos, Disemethate, Trichlorfor, Chlorothion are examples of other organic phosphorus insecticides.

DISEASE	ETOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD / SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
Chlorinated hydrocarbons.	Hydrocarbon Carbon Poison (Cutanea tarda 2-4-D, Methoxy- porphyrta) Hexachlorobenzene: Hexachlorobenzene, chlor, Chlor- benzilate. (75,124,166, 167,215,231)	Stimulates central nervous system. Liver poison. Fat soluble, insoluble in water. Pro-longed storage of DDT in mammals.	1/2 to 6 hours. Nausea, vomiting, paresthesia of tongue, lips, parts of face and extremities. Apprehension, disturbance of equilibrium, dizziness, confusion, muscular weakness, anorexia, weight loss. Inhaled vapor of Lindane: severe headache, nausea, irritation of eyes, nose, and throat.	Insecticides. Spray applications. Overheated thermal insecticide vaporizers. Hexachlorobenzene fungicide.	BHC: Seed grain, grain, bread made from treated grain, maternal milk. Toxaphene: leafy vegetables. Any food accidentally contaminated with insecticides.	Blood, urine, feces, fat biopsy, stomach contents. Extraction, Phenate ion formations, chromatography.	Avoid spraying just before harvest. Label and store insecticides in an area separate from food. Wash or blanch food. Restrict use of Lindane vaporizers and DDT.
Aldrin, Dieldrin, Isodrin, Endrin, Chlordane, Heptachlor.	Depresses central nervous system and irritates respiratory system.	Stimulates central nervous system.	1/2 hour or more. Similar to above but without paresthesia.	Insecticides.	Endrin: flour, bread, cola. Any food accidentally contaminated with insecticides.	Blood, urine, feces, fat biopsy, stomach contents.	Label and store in an area separate from food. Avoid storing insecticides in reusable containers.
D-D, Nemagon.			Soil fumigant.	Any food accidentally contaminated with fumigant.	Blood, urine, feces, fat		

DISEASE	ETOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD / SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS / LABORATORY	CONTROL MEASURES
Carbamate Poisoning (75,167,215)	Carbaryl (Sevin) Baygon (Propoxur) Motham, Temik (Aldicarb), Zectran	Reversible inhibitor of cholinesterase.	1/2 hour. Epigastric pain, vomiting, abnormal salivation, sweating, contraction of pupils, muscular incoordination.	Insecticides.	Cucumbers, flour roti (unleavened bread). Any food accidentally contaminated with insecticides.	Blood. Extraction, chromatography, colorimetry.	Label and store in an area separate from food.
Fluoride Poisoning (75,198, 255,290)	Sodium fluoride.	Fluorine is the most reactive chemical element. Direct cellular poison, interferes with calcium metabolism and enzyme mechanisms.	Few minutes to 2 hours. Salty or soapy taste, numbness of mouth, thirst, nausea, vomiting, diarrhea, abdominal burning and cramps, collapse, pallor, weakness, weak pulse, shallow respiration, wet and cold skin, cyanosis, dilated pupils, spasms, shock, death.	Roach and rat poison.	Contaminated scrambled eggs, flour, mistaken and used for baking soda, baking powder, and dry milk.	Food, vomitus, stomach contents. Ash, dissolve fluoride ion electrode.	Color code insecticides. Label and store roach powder in an area separate from food.
Sodium/ Monofluoro-acetate Poisoning (75,166,167)	Sodium monofluoroacetate (LD50).	Cardiovascular or nervous system toxin.	1/2 to 2 hours. Nausea, vomiting, mental apprehension, epileptiform convulsions, cardiac arrest or ventricular fibrillation, death may occur.	Rodenticide.	Any food accidentally contaminated with rodenticide.	Blood, organs. Prohibit use.	Wash, extract, chromatography, fluoride ion electrode, mass spectroscopy.
Thallium Poisoning (70,75,84, 166,167)	Thallium sulfate.	Cellular toxin.	12 to 24 hours. Gastroenteritis: abdominal pain, vomiting, diarrhea (bloody), anorexia, stomatitis, salivation, weight loss. Neurological and other symptoms: paresthesia, headache, cranial nerve damage, insomnia, convulsions, delirium, coma, vascular collapse and death may occur. Loss of hair.	Rodenticide.	Barley brain and any food accidentally contaminated with rodenticide.	Urine, blood, hair. Ash, dissolve atomic absorption spectrometry.	Restrict use. Label and store in an area separate from food.
Warfarin Poisoning (75,166,167)	Warfarin.	Anticoagulant, inhibits pro-thrombin formation, capillary damage.	7 to 10 days. Back and abdominal pain, vomiting, nose bleeds, bleeding gums, pallor, petechial rash, massive bruises, extensive blood loss (seen in urine and feces), shock.	Rodenticide. Consumption of poisoned bait.	Blood, urine stomach contents. Extract, chromatography, spectrophotofluorometry.	Blood, urine and store in an area separate from food. Vitamin K treatment.	Corn meal. Any food accidentally contaminated with this rodenticide and eaten over a period of several days.

DISEASE	ETOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Phosphide Poisoning (75,166,167, 264)	White or yellow phosphorus, zinc phosphide.	Hydrogen phosphide (phosphine) released when stomach hydrochloric acid reacts with phosphide or phosphorus. Gastro-intestinal irritant.	1/2 to 9 hours. Burning pain, thirst, nausea, vomiting, diarrhea, abdominal pain. Symptom-free stage, then protracted vomiting and diarrhea, hantemesis, jaundice, hepatomegaly, oliguria, convulsions, delirium, coma, shock, severe damage to liver, heart, and kidneys. Death may occur. Characteristic odor of garlic to breath and vomitus. Greenish-black lumenous sputum, vomitus, and feces.	Rodenticides, match heads.	Barley. Any food accidentally contaminated with this pesticide.	Vomitus, feces, Label and store in urine, blood. Urinalysis, liver function tests. Ash, dissolve, photometry.	Label and store in an area separate from food.
Barium Poisoning (70,75, 162,223)	Barium carbonate	Causes local irritation, peripheral vasoconstriction, digitalis-like action on heart, and paralysis of central nervous system.	1 to 8 hours. Excessive salivation, vomiting, abdominal cramps, diarrhea (watery and bloody), tingling sensation of face and neck, loss of tendon reflexes, twitching, disordered action of heart muscles, paralysis, weakness, collapse, respiratory difficulty and failure.	Rodenticide.	Flour, bread, pastry tarts, potato starch, sausage. Any food contaminated with this pesticide.	Urine, feces. Autopsy: stomach and bowel contents, liver, bone. Precipitate, colorimetry.	Label and store in an area separate from food.
Nicotine Poisoning (41,75)	Nicotine sulfate.	Alkaloid that is caustic. Stimulates and then depresses central nervous system. Cerebral convulsant. Carcinogenic.	Burning sensation in mouth, anxiety, excitement, salivation, nausea, vomiting (odor of stale nicotine), diarrhea, abdominal pain, headache, visual disturbances, confusion, weakness, collapse, coma, convulsions, respiratory failure, flaccidity of muscles.	Insecticide. Tobacco products.	Any food accidentally contaminated with this pesticide. Mustard, cigarette butts.	Urine, Autopsy: stomach contents, liver, kidney. Extract, distill, separate, spectrophotometry.	Label and store in an area separate from food.

DISEASE	ETOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Red Squill Poisoning	(See Glycocides, Plant Toxicants and Toxins, page 72)						
Strychnine Poisoning	(See Alkaloids, Plant Toxicants and Toxins, page 72)						
Epoxy Resin Poisoning (188)	4,4' - diamino-diphenyl-methane.	Aromatic amine polyester.	Abdominal pain, jaundice, fever, malaise, muscular pains.	Epoxy resin hardener. Spillage during transfer.	Cereal grains.	Liver biopsy.	Avoid storing and transporting resin or catalyst with food.
Calcium chloride Poisoning (269)	Calcium chloride.	Corrosive.	Few minutes. Nausea, vomiting.	Brine from freezer tank.	Popsicles.	Vomitus.	Properly install vending machine water lines. Prevent cross-contamination.
Chromium Poisoning (162,267)	Sodium dichromate, trisodium phosphate, and sodium hydroxide.	Irritating and corrosive.	15 minutes. Vomiting, severe abdominal cramps, anorexia.	Rust and corrosion preventive compound (vending machine hooked up to hot water line).	Soft drinks.	Vomitus, urine. Ash, spectrophotometry.	Properly install vending machine water lines. Prevent cross-contamination.
Cyanide Poisoning (75)	Compounds containing cyanide.	Corrosive in stomach. Inhibits cytochrome oxidase system for oxygen utilization in cells.	1/2 to 6 hours. Nausea, vomiting (peach pit odor), diarrhea. Death caused by asphyxiation.	Silver polish (questionable transmission from this source). Fumigant for rats.	Any contaminated food.	Blood, urine. AgNO_3 precipitation, titration.	Avoid use of silver polish containing cyanide for food contact surfaces. Store in area separate from food. Label.
Lye Poisoning (53,75,270)	Sodium hydroxide.	Caustic (high pH). Corrosive.	Pew minutes to 12 to 14 hours. Burning of mouth, nausea, vomiting, edema of pharynx and larynx, collapse, coma may occur with high concentrations. Abdominal pain, diarrhea with low concentrations.	Drain cleaners, paint removers, paint, putty, hair straighteners, cleaning agents, washing compounds in bottling plants.	Any contaminated food.	Vomitus, food. Measure pH; add As_2O_3 and titrate.	Label and store in an area separate from food. Avoid using food containers for storing lye.
Soap Poisoning	Soap	Caustic.	Few minutes. Burning of tongue, mouth, and vocal cords.	Soaps.	White wine bottles used to store soap solutions.	Vomitus.	Avoid storing soaps and detergent solutions in empty wine bottles or other food containers. Label and store in an area separate from foods.

DISEASE	ETIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Anionic Detergent Poisoning (53)	Anionic detergent	Irritant	Few minutes. Vomiting and diarrhea.	Detergents.	Empty wine or soft drink bottles used to store detergent solutions.	Vomitus.	Avoid storing soaps and detergents in empty wine or soft drink bottles or other food containers. Label and store in an area separate from food.
Cationic Detergent Poisoning (53)	Cationic detergent	Interferes with cellular functions	Few minutes. Nausea, vomiting, shock, coma, convulsions, death.	Sanitizers, detergents.	Empty wine or soft drink bottles used to store detergent solutions.	Vomitus. Separate, colorimetry, optical-crystallography.	Avoid storing soaps and detergents in empty wine or soft drink bottles or other food containers. Label and store in an area separate from food.
Methyl alcohol Poisoning (75,92)	Methyl alcohol	Methyl (wood) alcohol.	8 to 72 hours. Vomiting, severe abdominal pain, depression, weakness, headache, dimness of vision, dyspnea, coma, cyanosis. Cerebral edema, optic neuritis, blindness, oliguria.	Paint solvent, denaturant in rubbing alcohol, antifreeze.	Ethyl alcohol substitute. Bootleg whiskey.	Urine, vomitus, blood, tissues after autopsy.	Avoid drinking wood alcohol. Store in an area separate from food. Label and add dye.
Mercuric chloride - vinyl complex (see page 162,125,129, for more information about mercury poisoning.)	Mercuric chloride - vinyl complex.	Central nervous system involvement. Bacterial conversion of mercury to methyl mercury. Causes degenerative changes in kidneys.	1 week or longer. Progressive numbness of extremities, lips, and tongue; ataxic gait, weakness of legs, loss of motor coordination of hands, dysarthria, dysphagia, deafness, blurring of vision, blindness, spasticity, rigidity, insomnia, childlike facial expression, coma. High case fatality rate.	Factor waste pollution. Mercury complex picked up by marine organisms, vectored to fish without harm, but were lethal to man. Occurred in Japan. Fungicide used to treat seed grains.	Crab, shellfish, marine invertebrates, hair. Shell-fish, fish, water, mud, and organs.	Urine, feces, blood, tissues, hair. Shell-fish, fish, water, mud, and organs.	Avoid eating grains treated with mercury compounds. Avoid feeding mercury contaminated grains to animals which are used for human foods. Prevent industrial mercury waste pollution. Avoid eating fish from mercury-polluted water.

DISEASE	ETOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Itai Itai (Dutch Ouch Disease, Chronic Cadmium Poisoning) (75, 145, 162, 185)	Cadmium.	Toxic changes in proximal renal tubules, hypercalcifuric.	Several months. Prolonged Ingestion hypercalcuria (blood urine), extreme bone pain due to osteomalacia, lambgo, typical cries of pain, pain in back, shoulders, joints, waddling gait to inability to walk, fractures under strain, proteinuria and renal lesion, glycosuria, aminoaciduria.	Mining wastes deposited in rice paddies over many years. Middle-aged women deficient in calcium and with multiple pregnancies most susceptible.	Rice, soybeans.	Blood, urine, rice, soil. Urinalysis, ash, spectrophotometry.	Eat adequate protein diet. Avoid continued ingestion of foods grown in soil with high concentration of cadmium. Prevent mining waste pollution of agricultural land.
Yusho (Rice Oil Disease, PCB Poisoning) (169, 194)	Poly-chlorinated biphenyls.	Attacks liver and skin. Affects newborn infants of poisoned mothers.	Several months. Dark brown pigmentation of nails, acne-like eruptions, increased eye discharge, visual disturbances, pigmentary of skin, lips, gingiva, swelling of upper eyelids, hyperemia of conjunctiva, enlargement and elevation of hair follicles, itching, pruritis, increased sweating of palms, hyperkeratotic plaques on soles and palms, weakness. Recovery takes years.	Heat-transfer agent. Environmental pollutant from transformer, condenser, copy paper, recycling paper plants.	Salad oil.	Blood, skin, fatty tissue, oil. Extraction, chromatography. SGOT, SGPT	Construct heat-exchange equipment to prevent contamination of food. Prevent industrial waste pollution of water.
Toxic Pneumonia (207)	Unknown (cooking oil ingredient)	Causes acute respiratory toxicity	Interstitial pneumonia fever, dry cough, headache, dyspnea, chest pain, vomiting, variable rash, pruritis, diarrhea, obstruction, takes years.	Significant association with ingestion of vegetables dressed with raw oil; occurred in Spain.	Oil, a mixture of ripe seed oil, liquified pork fat, raw oil; quality oil.	Oil, x-ray patients, analysis for Toxins Gas chromatography, animal toxicity testing.	Purchase oil for salad dressing and cooking from reputable company. Ingredients on label.

DISEASE	EPILOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Soy Protein Extender Extender (defatted soy flakes--texturized vegetable protein.	Soy protein extender (defatted soy flakes--texturized vegetable protein.	Unknown	Immediate to 6 hours, usually 1 hours or less. Nausea, abdominal cramps, dizziness, diarrhea, headache, difficulty breathing, vomiting, facial flushing, metallic taste, itching, swelling of tongue, lips, or face.	Food additive.	Tuna fish and other salads	Blood, feces, urine, hair, coconut (<i>Lecythis ollorii</i>). Beer.	Increase time-temperature of heating extender during processing.
Selenoamino acids.	Selenoamino acids.		Dermatitis, fatigue, dizziness, nausea, vomiting, diarrhea, loss of hair, loss of nails, discoloration of skin, dental caries.	Selenium-acid soil: Plants absorb seleniferum. Beer containing selenic acid from impure sulphuric acid.	Home-grown food with high concentration of selenium. Monkey coconut (<i>Lecythis ollorii</i>). Beer.	Blood, feces, urine, hair, food, soil. Wet digestion, fluorometry.	Avoid eating food known to contain high concentration of selenium. Peel vegetables. Mill grains. Discard water after cooking.
Allergens or Enzyme Deficiencies							
Gastro-intestinal and Food Allergies (3,89, 154,230)	Allergens react with antibody and form histamine or histamine-like substances.	Usually protein, sometimes sugars or fats. Depends on quantity, duration during which food is eaten, and regularity with which eaten.	Usually 2 to 12 hours. Immediate: instantly to < 4 hours. Delayed: few hours to day or two. Gastrointestinal: Nausea, vomiting, abdominal cramps, diarrhea, constipation, bloating, excessive gas, backache. Urticaria, angioedema, itching, aphthous stomatitis; Swelling of tissues, edema, hives, rash, eczema or dilation of blood vessels, spasm of smooth muscles, asthma, rhinitis may occur instead of gastrointestinal symptoms.	Initial response to food antigen, latent period (sensitizing taking place and antibody production), release of histamine and symptoms. Dietary history important in diagnosis, elimination diet.	Milk (protein constituents) and milk products, eggs (whites), cereals (wheat, buckwheat, corn, rice, rye, oats), fish, nuts, seafood, meat, vegetables (celery, string beans, lima beans, tomatoes), fruits (oranges, strawberries, bananas, lemons, watermelons), preservatives.	Skin tests.	Avoid eating responsible food or reduce quantity eaten. Cooking may destroy allergen.

DISEASE *	ETOIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Di-saccharide Intolerance (59, 195)	Lactose, sucrose, or isomaltose.	Congenital defect of enzyme deficiency of disaccharidases causes defects in absorption of dietary disaccharides. Sugars ferment in colon.	Bloating, frothy diarrhea, flatulence, abdominal pain.	High incidence in blacks and orientals.	Milk and other foods containing disaccharides.	Stool (after ingestion of test sugar). Blood. Biopsy (perioral).	Avoid eating offending disaccharide. Withdrawal of milk from diet (lactose intolerance).
Favism (See Plant Toxicants and Toxins, page 49 for detailed description.)							
Food-Drug Combinations							
Amine Poisoning (96)	Tyramine (aromatic amine).	Tyramine in cheese can be degraded to p-hydroxyphenyl-acetic acid by monoamine oxidase inhibitors in certain tranquilizers causing hypertensive attacks.	10 minutes to 2 hours. Palpitation, severe throbbing headache, hypertension, flushing, profuse perspiration, neck stiffness, photophobia, nausea, vomiting, prostration. Cerebro-vascular accidents may occur. Duration 10 minutes to 6 hours.	Cheese, monoamine oxidase inhibiting drugs (Butonyl, Niamid, Marplan, Nardil, Parnate, Marsalid, Catron, Monase).	Cheese, canned milk, pickled herring, alcohol, cream, broad beans, yeast extract.	Distillation, extraction, spectrophotometry, chromatography.	Persons taking monoamine oxidase inhibiting drugs should avoid eating cheese and other listed food. Withdraw drugs.
Radioactive Substances							
Radio-nuclide Poisoning (120)	Various radio-nuclides, such as:	Symptoms depend on dosage, time, and organ affected. Genetic effect: lethal mutants, other mutations, shorter life span. Somatic effect: radiation sickness, leukemia, tumors, physical ailments.	Fallout, reactor plant accidents, radioactive wastes, natural substances.	Green, leafy legumes, vegetable tables, milk, fish, shellfish, meat, cereals.	Stop nuclear weapons testing, safe construction of reactor plants, monitor to evaluate hazard.	Separation, Gamma-ray spectroscopy, ion exchange.	Tissue of affected organ, foods potentially involved.

DISEASE	ETIOLOGIC AGENT	NATURE OF POISON	INCUBATION (LATENT) PERIOD/ SIGNS AND SYMPTOMS	SOURCE, RESERVOIR, AND EPIDEMIOLOGY	FOODS INVOLVED	SPECIMENS/ LABORATORY	CONTROL MEASURES
Strontium 89	β radiation. Half life 51 days.	Bone cancer and leukemia.	Atmosphere to soil or plant. Plant to cow to milk or milk products to man (bone).	Milk	Ion exchange.	Peel and wash produce.	
Strontium 90	β radiation. Half life 28 years.	Bone cancer and leukemia.	Atmosphere to soil or plant. Plant to cow to milk or milk products to man (bone).	Green, leafy vegetables, milk, and milk products.	Peel and wash produce. Ion exchange for liquid food. Remove bones from meat.		
Iodine 131	β , γ radiation. Half life 8.1 days.	Atmosphere to soil to cow to milk to man (thyroid).	Atmosphere to plant to cow to milk to man (thyroid).	Milk.	Convert milk to dairy product and store for several weeks - monitor.		
Cesium 137	β , γ radiation. Half life 30 years.	Atmosphere to soil or plant. Plant to cattle to milk, meat, or other animal product to man; or atmosphere to water to seed plants, plankton, sediment, and algae to crustaceans, shellfish, fish to man or to predatory fish to man (total body man (total body liver, spleen, muscle)).	Atmosphere to soil or plant. Plant to cattle to milk, meat, or other animal product to man; or atmosphere to water to seed plants, plankton, sediment, and algae to crustaceans, shellfish, fish to man or to predatory fish to man (total body man (total body liver, spleen, muscle)).	Green, leafy vegetables, milk, milk products, meat, shell-fish, fish.	Peel and wash produce.		
Phosphorus 32	β radiation. Half life 14.3 days.	Atmosphere to plant to cow to milk to man (bone).	Atmosphere to plant to cow to milk to man (bone).	Green, leafy vegetables.	Peel and wash produce. Store for several weeks - monitor.		
Barium 140	β , γ radiation. Half life 12.8 days. Sequestered by tissue.	Atmosphere to plant to cow to milk to man (bone).	Milk.	Atmosphere to plant to cow to milk to man (bone).	Convert milk to dairy product. Store for several weeks - monitor.		
Ruthenium 106				Waste water to water to seaweed (<i>Porphyrumbilobatis</i>) to laverbread to man (lower large intestine).	Use seaweed from different sources. Eat different kind of bread.		

GENERAL REFERENCES

Multiple Categories of Diseases

1. Acha, P. N. and G. Szyfred. 1980. Zoonoses and communicable diseases common to man and animals. Pan American Health Organization, Washington, D.C.
2. Benenson, A. S. (ed.). 1981. Control of communicable diseases in man, 13th ed. American Public Health Association, Washington, D.C.
3. Beeson, P. B., W. McDermott, J. B. Wyngaarden (eds.). 1979. Cecil-Loeb textbook of medicine, 15th ed. W. B. Saunders Co., Philadelphia.
4. Braude, A. E., C. E. Davis, and J. Fierer. 1981. Medical microbiology and infectious diseases. W. B. Saunders Co., Philadelphia.
5. Bryan, F. L., H. W. Anderson, R. K. Anderson, K. J. Baker, H. Matsuura, T. W. McKinley, R. C. Swanson, and E. C. D. Todd. 1976. Procedures to investigate foodborne illness, 3rd ed. International Association of Milk, Food, and Environmental Sanitarians, Inc., Ames, IA. 51p.
6. Dack, G. M. 1956. Food poisoning, 3rd ed. University of Chicago Press, Chicago.
7. Dewberry, E. B. 1959. Food poisoning: Food-borne infections and intoxication: Nature, history, and causation. Measures for prevention and control. Leonard Hill, London.
8. Eickhoff, T. C. (ed.). 1978. Practice of medicine. Vol. III. Bacterial diseases, rickettsial infections, and Vol. IV. Virus infections, parasitic infections. Harper and Row, Hagerstown, MD.
9. Graham, H. D. (ed.). 1980. Safety of foods, 2nd ed. AVI Publishing Co., Westport, CT.
10. Hobbs, B. C. and J. H. B. Christian. 1973. The microbiological safety of food. Academic Press, London and New York.
11. Hubbert, W. T., W. F. McCulloch, P. R. Schnurrenberger (eds.). 1975. Diseases transmitted from animals to man, 6th ed. Charles C. Thomas, Publisher, Springfield, IL.
12. Reimann, H. and F. L. Bryan (eds.). 1979. Food-borne infections and intoxications, 2nd ed. Academic Press, New York.
13. Tartakow, J. and J. H. Vorperian. 1981. Foodborne and waterborne diseases. Their epidemic characteristics. AVI Publishing Co., Westport, CT.
14. Wherle, P. F. and F. S. Top, Sr. (eds.). 1981. Communicable and infectious diseases, 9th ed. C. V. Mosby Co., St. Louis, MO.

Bacterial Diseases (1-14)

15. DeFigueiredo, M. P. and D. F. Splittstoesser (eds.). 1976. Food microbiology: Public health and spoilage aspects. AVI Publishing Co., Westport, CT.
16. DuPont, H. L. and L. K. Pickering. 1980. Infections of the gastrointestinal tract. Plenum Medical Book Co., New York.
17. Genigeorgis, C. A. and H. Riemann. 1973. Food safety and food poisoning. World Rev. Nutr. Dietet. 16:363-394.
18. Hobbs, B. C. and R. J. Gilbert. 1978. Food poisoning and food hygiene, 4th ed. Edward Arnold, Lond.
19. International Commission on Microorganisms in Foods. 1978. Microorganisms in foods. 1. Their significance and methods of enumeration. University of Toronto Press, Toronto.
20. Mossel, D. A. H. 1975. Microbiology of foods and dairy products: Occurrence, prevention and monitoring of hazards and deterioration. CRC Crit. Rev. Environ. Control 5:1-139.
21. Speck, M. L. 1976. Compendium of methods for the microbiological examination of foods. American Public Health Association, Washington, D.C.
22. Steele, J. H. (ed.). 1979. CRC handbook series in zoonoses. Vol. I. and Vol. II. CRC Press, Boca Raton, FL.
23. Tanner, F. W. and L. P. Tanner. 1953. Food-borne infections and intoxication, 2nd ed. Garrard Press, Champaign, IL.
24. Tu, A. T. (ed.). 1980. Survey of contemporary toxicology. Vol. I. John Wiley and Sons, New York.
25. Wilson, G. S. and A. A. Miles. 1975. Topley and Wilson's principles of bacteriology and immunity. Vols. I. and II., 6th ed. Williams and Wilkins, Baltimore.

Viral Diseases (3, 4, 8, 11, 12, 14)

26. Andrews, C., H. G. Pereira, and P. Wildy. 1978. Viruses of vertebrates, 4th ed. Baillière Tindall, London
27. Cliver, D. O. 1979. Viral infections. In: H. Reimann and F. L. Bryan (eds.). Food-borne infections and intoxications, 2nd ed. Academic Press, New York.
28. Evans, A. S. (ed.). 1976. Viral infections of humans. Epidemiology and control. Plenum Medical Book Co., New York.

29. Kurstak, E. and C. Kurstak. 1977. Comparative diagnosis of viral diseases, Vols. I. and II. Human and related viruses, Parts A and B. Academic Press, New York.
30. Lennette, E. H., and N. J. Schmidt (eds.). 1979. Diagnostic procedures for viral, rickettsial and chamydial infections, 5th ed. American Public Health Association, Washington, D.C.

Parasitic Diseases (3, 4, 8, 12, 14)

31. Faust, E. C., P. C. Beaver, and R. C. Jung. 1975. Animal agents and vectors of human disease, 4th ed. Lea and Febiger, Philadelphia.
32. Healy, G. R. and D. Juranek. 1979. Parasitic infections. In: H. Reimann and F. L. Bryan (eds.). Food-borne infections and intoxications, 2nd ed. Academic Press, New York.
33. Hunter, G. W., III, J. C. Swartzwelder, and D. F. Clyde. 1976. Tropical medicine, 5th ed. W. B. Saunders Co., Philadelphia.
34. Kean, B. H., K. E. Mott, and A. J. Russell. 1978. Tropical medicine and parasitology. Classic investigations. Vols. 1 and 2. Cornell University Press, Ithaca, NY.
35. Kreier, J. P. (ed.). 1977, 1978. Parasitic protozoa. Vol. II. Intestinal flagellates, histomonads, trichomonads, amoeba, opalinids, and ciliates. Vol. III. Gregarines, haemogregarines, coccidia, plasmodia, and haemoproteids. Academic Press, New York.
36. Manson-Bahr, P. E. C. and F. I. C. Apted. 1982. Manson's tropical diseases, 18th ed. Baillière Tindall, Cassell, London.
37. World Health Organization Expert Committee. 1979. Parasitic zoonoses. Tech. Rep. Ser. 637. World Health Organization, Geneva.

Fungal Diseases

38. Hardin, J. W. and J. M. Arena. 1974. Human poisoning from native and cultivated plants, 2nd ed. Duke University Press, Durham, NC.
39. Kadis, S., A. Ciegler, and S. J. Ajl (eds.). 1971-1972. Microbial toxins. Vol. VI. Fungal toxins. Vol. VII. Algal and fungal toxins. Vol. VIII. Fungal toxins. Academic Press, New York.
40. Lampe, K. F. 1979. Toxic fungi. Am. Rev. Pharmacol. Toxicol. 19:85-104.
41. Lampe, K. F. and R. Fagerström. 1968. Plant toxicity and dermatitis. Williams and Wilkins, Baltimore.
42. Lincoff, G. and D. H. Mitchel. 1977. Toxic and hallucinogenic mushroom poisoning. A handbook for physicians and mushroom hunters. Van Nostrand Reinhold Co., New York.

43. Marth, E. H. and B. C. Calonog. 1976. Toxigenic fungi. In: M. P. DeFigueiredo and D. F. Splitstoesser (eds.). *Food microbiology: Public health and spoilage aspects*. AVI Publishing Co., Westport, CT.
44. Rodricks, J. V., C. W. Hesseltine, and M. A. Mehlman (eds.). 1977. *Mycotoxins in human and animal health*. Pathotox Publishers, Park Forest South, IL.
45. Rumack, B. H. and E. Salzman (eds.). 1978. *Mushroom poisoning: Diagnosis and treatment*. CRC Press, West Palm Beach, FL.
46. Smith, A. H. 1949. *Mushrooms in their natural habitats*. Vols. I. and II. Sawyer, Portland, OR.
47. Tyler, V. E., Jr. 1963. *Poisonous mushrooms*. *Progr. Chem. Toxic.* 1:339-384.
48. Tyler, V. E., L. R. Brady, and J. E. Robbers. 1976. *Pharmacognosy*, 7th ed. Lea and Febiger, Philadelphia.
49. Uraguchi, K. 1971. *Pharmacology of mycotoxins*. In: H. Rásková (ed.). *Pharmacology and toxicology of naturally-occurring toxins*. Pergamon Press, Oxford.
50. Wilson, B. J. and A. W. Hayes. 1973. *Microbial toxins*. In: (Committee on Food Protection, National Research Council) *Toxicants occurring naturally in foods*. National Academy of Sciences, Washington, D.C.
51. Wogan, G. N. and W. Busby. 1979. *Alimentary mycotoxicoses*. In: H. Reimann and F. L. Bryan (eds.). *Food-borne infections and intoxications*, 2nd ed. Academic Press, New York.
52. Wyllie, T. D. and L. G. Morehouse (eds.). 1977. *Mycotoxic fungi, mycotoxins, mycotoxicoses*. An encyclopedic handbook. Vol. 1. *Mycotoxic fungi and chemistry of mycotoxins*. Vol. 2. *Mycotoxicoses of domestic and laboratory animals, poultry, and aquatic invertebrates*. Vol. 3. *Mycotoxicoses of man and plants: mycotoxin control and regulatory practices*. Marcel Dekker, New York.

Plant Toxicants and Toxins (38, 41, 48)

53. Arena, J. W. 1979. *Poisoning toxicology, symptoms, treatments*, 4th ed. Charles C. Thomas Publisher, Springfield, IL.
54. Duke, J. E. 1977. *Phytotoxin tables*. *CRC Crit. Rev. Toxicol.* 5:189-237.
55. Ellis, M. D. (ed.). 1978. *Dangerous plants, snakes, arthropods and marine life. Toxicity and treatment*. Drug Intelligence Publications. Hamilton, IL.
56. Kinghorn, A. D. (ed.). 1979. *Toxic plants*. Columbia University Press, New York.

57. Kingsbury, J. M. 1964. Poisonous plants of the United States and Canada. Prentice-Hall, Englewood Cliffs, NJ.
58. Lewis, W. H. and M. P. F. Elvin-Lewis. 1977. Medical botany: Plants affecting man's health. John Wiley and Sons, New York.
59. National Research Council, Committee on Food Protection. 1973. Toxicants occurring naturally in foods, 2nd ed. National Academy of Sciences, Washington, D. C.
60. Radeleff, R. D. 1970. Veterinary toxicology, 2nd ed. 1970. Lea and Febiger, Philadelphia.
61. Schwarting, A. E. 1963. Poisonous seeds and fruits. Progr. Chem. Toxic. 1:385-401.
62. Tampion, J. 1977. Dangerous plants. Universe Books, New York.
63. Trease, G. E. and W. C. Evans. 1978. Pharmacognosy, 11th ed. Baillière Tindall, London.
64. Watt, J. M. and M. G. Breyer-Brandwijk. 1962. The medicinal and poisonous plants of southern and eastern Africa. Being an account of their medicinal and other uses, chemical composition, pharmacological effects and toxicology in man and animals. E. and S. Livingstone, Edinburgh.

Toxic Animals (54, 58)

65. Halstead, B. W. 1978. Poisonous and venomous marine animals of the world, 2nd ed. Darwin Press, Princeton, NJ.
66. Liener, I. E. 1974. Toxic constituents of animal foodstuffs. Academic Press, New York.
67. Russell, F. E. 1971. Pharmacology of toxins of marine organisms. In: H. Rašková (ed.). Pharmacology and toxicology of naturally occurring toxins. Vol. II. Pergamon Press, Oxford.
68. Schantz, E. J. 1973. Seafood toxicants. In: (Committee on Food Protection, National Research Council). Toxicants occurring naturally in foods. National Academy of Sciences, Washington, D.C.
69. Taylor, D. L. and H. H. Seliger. 1979. Toxic dinoflagellate blooms. Elsevier/North Holland, New York.

Poisonous Chemicals (53)

70. Browning, E. 1969. Toxicity of industrial metals, 2nd ed. Appleton-Century-Crofts, New York.
71. Deichmann, W. B. and H. W. Gerarde. 1969. Toxicology of drugs and chemicals. Academic Press, New York.
72. Doull, J., C. D. Klaassen, and M. O. Amdur. 1980. Casarett and Doull's Toxicology. The basic science of poisoning, 2nd ed. Macmillan Publishing Co., New York.
73. Dreisbach, R. H. 1980. Handbook of poisoning: Prevention, diagnosis and treatment, 10th ed. Lang Medical Publications, Los Altos, CA.
74. Gilman, A. G., L. S. Goodman, and A. Gilman (eds.). 1980. Goodman and Gilman's the pharmacological basis of therapeutics, 6th ed. Macmillan Publishing Co., New York.
75. Gosselin, R. E., H. C. Hodge, R. P. Smith, and M. W. Gleason. 1976. Clinical toxicology of commercial products: Acute poisoning, 4th ed. Williams and Wilkins, Baltimore.
76. Horwitz, W. (ed.). 1980. Official methods of analysis of the Association of Official Analytical Chemists, 13th ed. Assoc. Official Analytical Chemists, Washington, D.C.
77. Lowry, W. T. and J. C. Garriott. 1979. Forensic toxicology. Controlled substances and dangerous drugs. Plenum Press, New York.
78. Sittig, M. 1981 Handbook of toxic and hazardous chemicals. Noyes Publications, Park Ridge, NJ.
79. Sollmann, T. 1957. A manual of pharmacology, 8th ed. W. B. Saunders Co., Philadelphia.
80. Venugopal, B. and T. D. Luckey. 1979. Metal toxicity in mammals, 2. Plenum Press, New York.

SPECIFIC REFERENCES

81. Albach, R. A. and T. Booden. 1978. Amoeba. In: J. P. Kreier (ed.). Parasitic Protozoa. Vol. II. Intestinal flagellates, histomonads, trichomonads, amoeba, opalinids, and ciliates. Academic Press, New York.
82. Alicata, J. E. and K. Jindrak. 1970. Angiostrongylosis in the Pacific and Southeast Asia. Charles C. Thomas, Publisher, Springfield, IL.
83. Arnold, S. H. and W. D. Brown. 1978. Histamine toxicity from fish products. Adv. Food Res. 24:113-154.

84. Banks, W. J., D. E. Pleasure, and K. Suzuki. 1972. Thallium poisoning. *Arch. Neurol.* 26:456-464.
85. Barker, W. H., Jr. 1974. *Vibrio parahaemolyticus* outbreaks in the United States. *Lancet* 1:551-554.
86. Barker, E. H., Jr. and V. Runte. 1972. Tomato juice associated gastroenteritis, Washington and Oregon, 1969. *Am. J. Epidemiol.* 96:219-226.
87. Barrett, F. F. and E. O. Mason. 1975. Diphtheria. In: T. C. Eickhoff (ed.). *Practice of medicine*. Vol. III. *Bacterial diseases, rickettsial infections*. Harper and Row, Hagerstown, MD.
88. Barua, D. and W. Burrows (eds.). 1974. Cholera. W. B. Saunders Co., Philadelphia.
89. Benack, R. T. 1967. What is allergy? A guide for the allergic person. Charles C. Thomas, Publisher, Springfield, IL.
90. Benedict, R. G. 1972. Mushroom toxins other than Aminita. In: S. Kadis, A. Ciegler, and S. J. Ajl (eds.). *Microbial toxins*. VIII. *Fungal toxins*. Academic Press, New York.
91. Bengtsson, E., L. Hassler, P. Holtenius, F. Nordbring, and G. Thoren. 1968. Infestation with *Dicrocoelium dendriticum*--the small liver fluke--in animals and human individuals in Sweden. *Acta Microbiol. Scandinav.* 74:85-92.
92. Bennett, I. L., F. H. Cary, G. L. Mitchell, and M. N. Cooper. 1953. Acute methyl alcohol poisoning: A review based on experiences in an outbreak of 323 cases. *Med.* 32:431-463.
93. Berde, B. and H. O. Schild. 1978. Ergot alkaloids and related compounds. Springer-Verlag, Berlin.
94. Bergdoll, M. S. 1979. Staphylococcal intoxication. In: H. Reimann and F. L. Bryan (eds.). *Foodborne infections and intoxications*, 2nd ed. Academic Press, New York.
95. Bhat, R. V., V. Nagarajan, and P. G. Tulpule. 1978. Health hazards of mycotoxins in India. Indian Council of Medical Research, New Delhi.
96. Blackwell, B., E. Marley, J. Price, and D. Taylor. 1967. Hypertensive interactions between monoamine oxidase inhibitors and foodstuffs. *Brit. J. Psychiat.* 113:349-365.
97. Black, P. A., M. H. Merson, R. E. Weaver, D. G. Hollis, and P. C. Heublein. 1979. Disease caused by a marine *Vibrio*: clinical characteristics and epidemiology. *N. Engl. J. Med.* 300:1-5.
98. Blake, P. A., R. E. Weaver, and D. G. Hollis. 1980. Diseases of humans (other than cholera) caused by *Vibrios*. *Ann. Rev. Microbiol.* 34:341-367.

99. Blazer, M. J., I. D. Berkowitz, M. LaForce, J. Cravens, B. Reller, and W. L. Wang. 1979. *Campylobacter enteritis*: Clinical and epidemiologic features. Ann. Intern. Med. 91:179-185.
100. Bodian, D. and D. M. Horstmann. 1965. Polioviruses. In: F. L. Horsfall and I. Tamm (eds.). *Viral and rickettsial infections of man*, 4th ed. J. B. Lippincott Co., Philadelphia.
101. Boray, J. C. 1969. Experimental *Fascioliasis* in Australia. Adv. Parasitol. 7:95-210.
102. Bottone, E. J. (ed.). 1981. *Yersinia enterocolitica*. CRC Press, Boca Raton, FL.
103. Bowman, W. C. and I. S. Sanghui. 1963. Pharmacological action of hemlock (*Conium maculatum*) alkaloids. J. Pharm. Pharmacol. 15:1-25.
104. Bove, F. J. 1970. The story of ergot. Karger, Basel.
105. Brachman, P. S. 1977. Anthrax. In: T. C. Eickhoff (ed.). *Practice of medicine*. Vol. III. *Bacterial diseases, rickettsial infections*. 1978. Harper and Row, Hagerstown, MD.
106. Breese, B. B. and C. B. Hall, 1978. Beta hemolytic streptococcal disease. Houghton Mifflin Professional Publishers, Boston.
107. Brown, M. A., J. V. Thom, G. L. Orth, P. Cova, and J. Juarez. 1964. Food poisoning containing zinc contamination. Arch. Env. Health 8:657-660.
108. Brown, P. 1980. An epidemiologic critique of Creutzfeldt-Jakob disease. Epidemiol. Rev. 2:113-135.
109. Bryan, F. L. 1976. *Staphylococcus aureus*. In: M. P. DeFigueiredo and D. F. Splitstoesser (eds.). *Food Microbiology: Public health and spoilage aspects*. AVI Publishing Co., Westport, CT.
110. Bryan, F. L. 1979. Infections and intoxications due to other bacteria. In: H. Reimann and F. L. Bryan (eds.). *Food-borne infections and intoxications*, 2nd ed. Academic Press, New York.
111. Bryan, F. L., H. Reimann, and M. J. Fanelli. 1979. *Salmonella* infections. In: H. Reimann and F. L. Bryan (eds.). *Food-borne infections and intoxications*, 2nd ed. Academic Press, New York.
112. Budd, W. 1977 (reprinted). *Typhoid its nature, mode of spreading, and prevention*. Arno Press, New York.
113. Butzler, J. P. and M. B. Skirrow. 1979. *Campylobacter enteritis*. Clin. Gastroenterol. 8:737-765.

114. Casals, J. 1976. Arenaviruses. In: A. S. Evans (ed.). *Viral infections of humans*. Plenum Medical Book Co., New York.
115. Casals, J. and S. M. Buckley. 1974. Lassa fever. *Progr. Med. Virol.* 18:111-126.
116. Cates, W., Jr., H. D. Silsby, and C. L. McFarlane. 1974. Diphenylhydantoin intoxication in military aviators--Germany. *Morbidity and Mortality Weekly Rpt.* 23(5):38-39.
117. Chapman, J. S. 1977. *The atypical mycobacteria and human mycobacteriosis*. Plenum Medical Book Co., New York.
118. Christensen, W. I. 1965. Milk sickness: A review of the literature. *Econ. Botany* 29:3-300.
119. Cohen, J. O. (ed.). 1972. *The staphylococci*. Wiley-Interscience, New York.
120. Committee on Food Protection, Food and Nutrition Board, National Research Council. 1973. Radionuclides in foods. National Academy of Sciences, Washington, D. C.
121. Committee on Medical and Biologic Effects of Environmental Pollutants, National Research Council. 1976. Selenium. National Academy of Sciences, Washington, D. C.
122. Committee on Medical and Biologic Effects of Environmental Pollutants, National Research Council. 1977. Copper. National Academy of Sciences, Washington, D. C.
123. Committee on *Salmonella*, National Research Council. 1969. An evaluation of the *Salmonella* problem. National Academy of Sciences, Washington, D.C.
124. Courtney, K. D. 1979. Hexachlorobenzene (HCB): A review. *Environ. Res.* 20:225-266.
125. Curley, A., V. A. Sedlak, E. F. Girling, R. E. Hawk, W. F. Barthel, P. E. Pierce, and W. H. Likosky. 1971. Organic mercury identified as the cause of poisoning in humans and hogs. *Sci.* 172:65-67.
126. Dalldorf, G. and J. L. Melnick. 1965. Coxsackie viruses. In: F. L. Horsfall and I. Tamm (eds.). *Viral and rickettsial infections of man*, 4th ed. J. B. Lippincott Co., Philadelphia.
127. Davis, B. R., G. R. Fanning, J. M. Madden, A. G. Steigerwalt, H. B. Bradford, Jr., H. L. Smith, Jr., and D. J. Brenner. 1981. Characterization of biochemically atypical *Vibrio cholerae* strains and designation of a new pathogenic species *Vibrio mimicus*. *J. Clin. Microbiol.* 14:631-639.

128. Dawes, B. and D. L. Hughes. 1964. *Fascioliasis: The invasive stages of Fasciola hepatica in mammalian hosts.* Adv. Parasitol. 2:97-168.
129. D'itri, P. A. and F. M. D'itri. 1977. Mercury contamination. A human tragedy. John Wiley and Sons, New York.
130. Doeglas, H. M. G., E. H. Hermans, and J. Huisman. 1961. The margarine disease. Arch. Dermatol. 83:837-843.
131. Doggett, R. E. (ed.). 1979. *Pseudomonas aeruginosa. Clinical manifestations of infection and current therapy.* Academic Press, New York.
132. Dubey, J. P. 1977. *Toxoplasma, Hammondia, Besnoitia, Sarcocystis,* and other tissue cyst-forming coccidia of man and animals. In: J. P. Kreier (ed.). *Parasitic protozoa.* Vol. III. Gregarines, Haemegregarines, Coccidia, Plasmodia, and Haemoproteids. Academic Press, New York.
133. Duncan, C. 1976. *Clostridium perfringens.* In: M. P. DeFigueiredo and D. F. Splittstoesser (eds.). *Food microbiology: Public health and spoilage aspects.* AVI Publishing Co., Westport, CT.
134. Edwards, P. R. and W. H. Ewing. 1972. *Identification of Enterobacteriaceae,* 3rd ed. Burgess, Minneapolis.
135. Edwards, P.-R., M. A. Fife, and C. H. Ramsey. 1959. Studies on the Arizona group of enterobacteriaceae. Bacteriol. Rev. 23:155-174.
136. Elling, F. 1977. Morphological aspects of mycotoxic nephropathy. In: J. V. Rodricks, C. W. Hesseltine, and M. A. Mehlman (eds.). *Mycotoxins in human and animal health.* Pathotox Publishers, Park Forest South, IL.
137. Elsdon-Dew, R. 1968. The epidemiology of amoebiasis. Adv. Parasitol. 6:1-62.
138. Everett, E. D. 1974. *Balantidiasis, giardiasis, and coccidiosis.* In: T. C. Eickhoff (ed.). *Practice of medicine.* Vol. III. Bacterial diseases, rickettsial infections, and Vol. IV. Virus infections, parasitic infections. Harper and Row, Hagerstown, MD.
139. Ewing, W. H., A. C. McWhorter, M. M. Ball, and S. F. Barles. 1969. *Edwardsiella tarda: Biochemical reactions.* J. Conf. Public Health Lab. Dir. 27:129-141.
140. Faber, H. K. and E. B. Shaw. 1977. In: T. C. Eickhoff (ed.). *Practice of medicine.* Vol. III. Bacterial diseases, rickettsial infections, and Vol. IV. Virus infections, parasitic infections. Harper and Row, Hagerstown, M.D.

141. Fassett, D. W. 1966. Nitrates and nitrates. In: Toxicants occurring naturally in foods. National Academy of Sciences Publ. No. 1354.
142. Feldman, H. A. 1974. Toxoplasmosis. In: T. C. Eickhoff (ed.) Practice of medicine. Vol. IV. Virus infections, parasitic infections. 1978. Harper and Row, Hagerstown, MD.
143. Francis, D. P. and J. E. Maynard. 1979. The transmission of hepatitis A, B, and non-A, non-B: A review. Epidemiol. Rev. 1:17-31.
144. Francis, J. 1958. Tuberculosis in animals and man. A study in comparative pathology. Cassell, London.
145. Friberg, L., M. Piscator, G. F. Nordberg, and T. Kjellstrom. 1974. Cadmium in the environment, 2nd ed. CRC Press, Cleveland.
146. Fuhrman, F. A. 1974. Fish eggs. In: I. E. Lienc (ed.). Toxic constituents of animal feedstuffs. Academic Press, New York.
147. Gajdusek, D. C. 1953. Acute infectious hemorrhagic fevers and myco-toxicoses in the Union of Soviet Socialist Republic. Medical Sci. Publ. 2. Walter Reed Army Medical Center, Washington, D. C.
148. Gajdusek, D. C. 1963. Kuru. Trans. Roy. Soc. Trop. Med. Hyg. 57:151-169.
149. Gajdusek, D. C. and C. J. Gibbs, Jr. 1975. Kuru, Creutzfeldt-Jakob disease, and transmissible presenile dementias. In: V. ter Meulen and M. Katz (eds.). Slow virus infections of the central nervous system. Springer-Vertag, New York.
150. Gangarosa, E. J. 1976. Asiatic cholera. In: T. C. Eickhoff (ed.). Practice of medicine. Vol. III. Bacterial infections. 1978. Harper and Row, Hagerstown, MD.
151. Gilbert, R. 1979. *Bacillus cereus* gastroenteritis. In: H. Reimann and F. L. Bryan (eds.). Food-borne infections and intoxications, 2nd ed. 1979. Academic Press, New York.
152. Gilbert, R. J., P. C. B. Turnbull, J. M. Parry, and J. M. Kramer. 1979. Food poisoning and other clinical infections associated with *Bacillus* species with particular reference to *B. cereus*. Symp. *Bacillus* spp. Soc. General Microbiology, London.
153. Ginsberg, H. S. and J. H. Dingle. 1965. The adenovirus group. In: F. L. Horsfall and L Tamm (eds.). Viral and rickettsial infections in man, 4th ed. 197 . J. B. Lippincott Co., Philadelphia.
154. Gleich, G. J., M. I. Sacks, and E. O. O'Connell. 1980. Hypersensitivity reactions induced by foods. In: C. W. Parker (ed.). Clinical immunology. Vol. 2. W. B. Saunders Co., Philadelphia.

155. Goepfert, J. M., W. M. Spira, and H. U. Kim. 1972. *Bacillus cereus*: Food poisoning organism. A review. *J. Milk Food Technol.* 35:213-227.
156. Goldblatt, L. A. 1969. *Aflatoxin*. Scientific background, control and implications. Academic Press, New York.
157. Gould, S. E. 1970. *Trichinosis in man and animals*. Charles C. Thomas, Publisher, Springfield, IL.
158. Griffin, R. B. and J. H. Knelson. 1975. *Lead*. Georg Thieme Publishers, Stuttgart (Academic Press, New York).
159. Griswold, D. M. 1950. Food poisoning: A review of thirty-four outbreaks. *Am. J. Public Health* 40:1398-1401.
160. Groger, D. 1972. Ergot. In: S. Kadis, A. Ciegler, and S. J. Ajl (eds.). *Microbiol toxins*. Vol. VIII. *Fungal toxins*. Academic Press, New York.
161. Gunn, R. A., P. R. Taylor, and E. J. Gangarosa. 1980. Gastrointestinal illness associated with consumption of a soy protein extender. *J. Food Prot.* 43:525-527.
162. Hammond, P. R. and R. P. Beliles. 1980. Metals. In: J. Doull, C. D. Klaassen, and M. O. Amdur (eds.). *Casarett and Doull's toxicology. The basic science of poisoning*, 2nd ed. Macmillan Publishing, New York.
163. Hart, M. 1963. Hazards to health: Jequirity bean poisoning. *New Engl. J. Med.* 268:885-886.
164. Hashimoto, Y., N. Fusetani, and S. Kimura. 1969. Aluterin: A toxin of filefish, *Alutera scripto*, probably originating from a zoantharin, *Polythoa tuberculosa*. *Bull. Japan. Soc. Sci. Fisheries* 35:1086-1093.
165. Hauschild, A. H. W. 1971. *Clostridium perfringens enterotoxin*. *J. Milk Food Technol.* 34:596-599.
166. Hayes, W. J., Jr. 1975. *Toxicology of pesticides*. Waverly Press, Baltimore.
167. Hayes, W. J., Jr. 1981. Pesticides studied in man. Williams and Wilkins, Baltimore.
168. Hickman, F. W., J. J. Farmer, III, D. G. Hollis, G. R. Fanning, G. R. Steigerwalt, R. E. Weaver, D. J. Brenner. 1982. Identification of *Vibrio hollisae* sp. nov. from patients with diarrhea. *J. Clin Microbiol.* 15:395-401.
169. Higuchi, K. (ed.). 1976. *PCB poisoning and pollution*. Kodansha, Toyko (Academic Press, New York).
170. Hirono, I. 1981. Natural carcinogenic products of plant origin. *CRC Critical Rev. Toxicol.* 8:235-277.

171. Hill, H. R. 1975. Streptococcal diseases. In: T. C. Eickhoff (ed.). Practice of medicine, Vol. III. Bacterial diseases, rickettsial infections. Harper and Row, Hagerstown, MD.
172. Hobbs, B. C. 1979. *Clostridium perfringens* infection. In: H. Reimann and F. L. Bryan (eds.). Food-borne infections and intoxications, 2nd ed. Academic Press, New York.
173. Hobbs, B. C., M. E. Smith, C. L. Oakley, and G. H. Warrack. 1953. *Clostridium welchii* food poisoning. J. Hyg. 51:75-101.
174. Hobbs, G. 1976. *Clostridium botulinum* and its importance in fishery products. Adv. Food Res. 22:135-185.
175. Hodgkinson, A. 1977. Oxalic acid in biology and medicine. Academic Press, London.
176. Hornick, R. B. and M. B. Gregg. 1975. Typhoid fever. In: T. C. Eickhoff (ed.). Practice of medicine, Vol. III. Bacterial diseases, rickettsial infections. 1978. Harper and Row, Hagerstown, MD.
177. Jacobs, L. 1973. New knowledge of toxoplasma and toxoplasmosis. Adv. Parasitol. 11:631-669.
178. Jeghers, H. 1943. Skin changes of nutritional origin. New Engl J. Med. 228:678-686.
179. Joffe, A. Z. 1971. Alimentary toxic aleukia. In: S. Kadis, A. Ciegler, and S. J. Ajl (eds.). Microbial toxins. Vol. VII. Algal and fungal toxins. Academic Press, New York.
180. Joffe, A. Z. 1978. *Fusarium poae* and *F. sporotrichioides* as principal causal agents of alimentary toxic aleuvia. In: T. D. Wyllie and L. G. Morehouse (eds.). Mycotoxic fungi, mycotoxins, mycotoxicoses. An encyclopedic handbook. Vol. 1. Mycotoxic fungi and chemistry of mycotoxins. Vol. 2. Mycotoxicoses of domestic and laboratory animals, poultry, and aquatic invertebrates and vertebrates. Vol. 3. Mycotoxicoses of man and plants: mycotoxin control and regulatory practices. Marcel Dekker, New York.
181. Johnson, K. M., S. B. Halstead, and S. N. Cohen. 1967. Hemorrhagic fevers of Southeast Asia and South America: A comparative appraisal. Prog. Med. Virol. 9:105-158.
182. Karunakaran, C. O. 1958. The Kerala food poisoning. J. Indian Med. Assoc. 31:204-207.
183. Kauffman, F. 1972. Serological diagnosis of salmonella-species. Kauffman-White-Schema. Williams and Wilkins, Baltimore.

184. Kinosita, R. and T. Shikata. 1964. On toxic moldy rice. In: G. N. Wogan (ed.). *Mycotoxins in food stuffs*. MIT Press, Cambridge, MA.
185. Kobayashi, J. 1970. Relation between the itai-itae disease and the pollution of river water by cadmium from a mine. *Adv. Water Pollution Res.* I(25):1-7.
186. Koff, R. S. 1978. *Viral hepatitis*. John Wiley and Sons, New York.
187. Komiya, Y. 1966. *Clonorchis* and clonorchiasis. *Adv. Parasitol.* 4:53-106.
188. Kopelman, H., M. H. Robertson, P. G. Sanders, and I. Ash. 1966. The Epping jaundice. *Brit. Med. J.* 1:514-516.
189. Krishnamachari, K. A. V. R., R. V. Bhat, V. Nagarjan, T. B. G. Tilak. 1975. Investigations into an outbreak of hepatitis in parts of Western India. *Indian J. Med. Res.* 63:1036-1049.
190. Krugman, S. and D. E. Gocke. 1978. *Viral hepatitis*. W. B. Saunders Company, Philadelphia.
191. Krogh, P. 1977. Ochratoxins. In: J. V. Rodricks, C. W. Hesseltine, and M. A. Mehlman (eds.). *Mycotoxins in human and animal health*. Pathotox Publishers, Park Forest South, IL.
192. [Kumamoto] Study Group on Minamata Disease. 1968. Minamata disease. Kumamoto Univ., Japan.
193. Kulda, J. and Nohyakova. 1978. Flagellates of the human intestine and of intestines of other species. In: J. P. Kreier (ed.). *Parasitic protozoa*. Vol. II. *Intestinal flagellates, histomonads, trichomonads, amoeba, opalinids, and ciliates*. Academic Press, New York.
194. Kuratsune, M., T. Yoshimura, J. Matsuzaka, and A. Yamaguchi. 1971. Yusho, a poisoning caused by rice oil contaminated with polychlorinated biphenyls. *HSMHA Health Reports* 86:1083-1091.
195. Lebenthal, E. 1978. Lactose intolerance. In: E. Lebenthal (ed.). *Digestive diseases in children*. Grune and Stratton, New York.
196. Lee, J. V., P. Shread, A. L. Furniss, and T. N. Bryant. 1981. Taxonomy and description of *Vibrio fluvialis* sp. nov. (Synonym Group F Vibrios, Group EF6). *J. Appl. Bacteriol.* 50:73-94.
197. Leedom, J. M. 1974. Q fever. In: T. C. Eickhoff (ed.). *Practice of medicine*. Vol. III. *Bacterial diseases, rickettsial infections*. Harper and Row, Hagerstown, MD.
198. Lidbeck, W. L., I. B. Hill, and J. A. Beeman. 1943. Acute sodium fluoride poisoning. *J. Am. Med. Assoc.* 121:826-827.

199. Lowry, W. T. and J. C. Garriott. 1979. *Forensic toxicology. Controlled substances and dangerous drugs.* Plenum Press, New York.
200. Mayer, C. F. 1953. Endemic panmyelotoxicosis in the Russian grain belt. Part I. The clinical aspects of alimentary toxic aleukia (ATA). A comprehensive review; Part II. The botany, phytopathology, and toxicology of Russian cereal feed. *Mil. Surg.* 113:173-189; 295-315.
201. McCabe, W. R. 1970. Trichinosis. In: T. C. Eickhoff (ed.). *Practice of medicine. Vol. IV. Virus infections, parasitic infections.* Harper and Row, Hagerstown, MD.
202. McDermott, P. H., R. L. Delaney, J. D. Egan, and J. F. Sullivan. 1966. Myocarditis and cardiac failure in men. *J. Am. Med. Assoc.* 198:163-166.
203. Meyer, E. A. and S. Radulescu. 1979. Giardia and giardiasis. *Adv. Parasitol.* 17:1-47.
204. Meyer, K. F. and B. Eddie. 1965. Sixty-five years of human botulism in the United States and Canada. Hooper Foundation, University of California, San Francisco.
205. Meyers, J. A. and J. H. Steele. 1969. *Bovine tuberculosis control in man and animals.* W. H. Green, St. Louis.
206. Middleton, P. J. 1977. Rotavirus: clinical observations and diagnosis of gastroenteritis. In: E. Kurstak and C. Kurstak (eds.). *Comparative diagnosis of viral diseases.* Academic Press, New York.
207. Ministerio de Sanidad y Consumo, Madrid, Spain and Centers for Disease Control. 1982. Follow-up on epidemic pneumonia with progression to neuromuscular illness. *Morbidity and Mortality Weekly Rpt.* 31(8):93-95.
208. Minor, T. E. and E. H. Marth. 1976. *Staphylococci and their significance in foods.* Elsevier Scientific Publishing Co., Amsterdam.
209. Miwatani, T., and Y. Takeda. 1976. *Vibrio parahaemolyticus - a causative bacterium of food poisoning.* Saikou Publishing Co., Tokyo.
210. Monath, T. P. 1975. Lassa fever: review of epidemiology and epizootiology. *Bull. World Health Org.* 52:577-592.
211. Morin, Y. L., A. R. Foley, G. Martinean, J. Russell, G. Mercier, G. Patry, G. Nadeau, H. Milon, A. Tetu, M. Samson, A. Lapointe, J. L. Bonenfant, G. Miller, P. E. Roy, C. Auger, J. Chenard, A. U. Sargent, B. Rose, P. K. Taskar, N. Senecal, and P. Daniel. 1967. Quebec beer-drinkers' cardiomyopathy. *Can. Med. Assoc. J.* 15:881-928.
212. Morton, J. F. 1970. Tentative correlations of plant usage and esophageal cancer zones. *Econ. Bot.* 24:217.
213. Morris, G. K. and J. C. Feeley. 1976. *Yersinia enterocolitica.* A review of its role in food hygiene. *Bull. World Health Org.* 54:179-185.

214. Mosely, J. W. 1975. The epidemiology of viral hepatitis: an overview. *Am. J. Med. Sci.* 270:253-270.
215. Murphy, S. D. 1980. Pesticides. In: J. Doull, C. D. Klaassen, and M. O. Amdur (eds.). *Casarett and Doull's toxicology. The basic science of poisoning*, 2nd ed. Macmillan Publishing, New York.
216. Murrell, T. G. C., J. R. Egerton, A. Rampling, J. Samels, and P. D. Walker. 1966. The ecology and epidemiology of pig-bel syndrome in man in New Guinea. *J. Hyg.* 64:275-296.
217. Muth, O. H. (ed.). 1967. *Symposium: selenium in biomedicine*. AVI Publishing Co., Westport, CT.
218. Narasimhan, M. J., Jr. 1971. Mycotoxic diseases in man and animals. *Intern. Symp. Phytopath. (Mycotoxins)*, New Delhi.
219. Narasimhan, M. J., Jr., V. G. Ganla, N. W. Deodhar, and C. R. Sule. 1967. Epidemic polyuria in man caused by a phycomycetous fungus (The Sassoon Hospital Syndrome). *Lancet* 1:760-761.
220. Nesterov, A. I. 1964. The clinical course of Kashin-Beck disease. *Arthritis Rheum.* 7:29-40.
221. Nilehn, B. 1969. Studies on *Yersinia enterocolitica* with special reference to bacterial diagnosis and occurrence in human acute enteric disease. *Acta Pathol. Microbiol. Scand. Suppl.* 206:5-48.
222. Noah, N. D., A. E. Bender, G. B. Raidi, and R. J. Gilbert. 1980. Food poisoning from red kidney beans. *Brit. Med. J.* 281:236-237.
223. Ogen, S., S. Rosenbluth, and A. Eisenberg. 1967. Food poisoning due to barium carbonate in sausage. *Israel J. Med. Sci.* 3:565-568.
224. Ouzounellis, T. 1970. Some notes on quail poisoning. *J. Am. Med. Assoc.* 211:1186-1187.
225. Padilla y Padilla, C. A. and G. M. Padilly (eds.). 1974. *Amebiasis in man. Epidemiology, therapeutics, clinical considerations, and prophylaxis*. Charles C. Thomas, Publisher, Springfield, IL.
226. Palmer, D. L. 1975. Amebiasis. In: T. C. Eickhoff (ed.). *Practice of medicine. Vol. IV. Virus infections, parasitic infections*. Harper and Row, Hagerstown, MD.
227. Paul, A. H. 1966. Chemical food poisoning by potassium bromate - report of an outbreak. *New Zealand Med. J.* 65:33-36.
228. Pawlowski, Z. and M. G. Schultz. 1972. Taeniasis and cysticercosis (*Taenia saginata*). *Adv. Parasitol.* 10:269-343.

229. Payne, R. B. 1963. Nutmeg intoxication. *New Engl. J. Med.* 269:36-38.
230. Perlman, F. 1977. Food allergens. In: N. Castimpoolas (ed.). *Immunological aspects of foods*. AVI Publishing Co., Westport, CT.
231. Peters, H. A. 1976. Hexachlorobenzene poisoning in turkey. *Food Proc.* 35:2400-2403.
232. Pollitzer, R. 1959. Cholera. World Health Organization, Geneva.
233. Press, E. and L. Yeager. 1962. Food poisoning due to sodium nicotinate. *Am. J. Public Health* 52:1720-1728.
234. Public Health Laboratory Service. 1976. Unusual outbreak of food poisoning. *Brit. Med. J.* 2:1268.
235. Ray, S. M. 1970. Current status of paralytic shellfish poisoning. In: *Proceedings SOS/70 Third Intern. Cong. Food Science and Technology*. Institute of Food Technologists, Chicago.
236. Reilly, J. and P. Tournier. Les Pasteurelloses Humanines. *Rev. Prat.* 4:1929-1937.
237. Rhodes, A. J. and C. E. Van Rooyen. 1968. *Textbook of virology*, 5th ed. Williams and Wilkins, Baltimore.
238. Rosen, L. 1968. Reoviruses. *Virol. Monog.* 1:73-107.
239. Rubin, R. H. and L. Weinstein. 1977. *Salmonellosis. Microbiologic, pathologic, and clinical features*. Stratton Intercontinental Medical Book Corp., New York.
240. Sack, B. 1975. Human diarrheal disease caused by enterotoxigenic *Escherichia coli*. *Ann. Rev. Microbiol.* 29:333-353.
241. Saito, M., and T. Tatsuno. 1971. Toxins of *Fusarium nivale*. In: S. Kadis, A. Ciegler, and S. J. Ajl (eds.). *Microbiol toxins*. Vol. VII. Algal and fungal toxins. Academic Press, New York.
242. Sakazaki, R. 1979. Vibrio infections. In: H. Reimann and F. L. Bryan (eds.). *Food-borne infections and intoxications*. Academic Press, New York.
243. Sakiguchi, G. 1979. Botulism. In: H. Reimann and F. L. Bryan (eds.). *Food-borne infections and intoxications*. Academic Press, New York.
244. Sanders, C. V. 1977. Tularemia. In: T. C. Eickhoff (ed.). *Practice of medicine*. Vol. III. *Bacterial diseases, rickettsial infections*. Harper and Row, Hagerstown, MD.
245. Sandmeyer, E. E. and C. J. Kirwin, Jr. 1981. Esters. In: G. D. Clayton and F. E. Clayton (eds.). *Patty's industrial hygiene and toxicology*, 3rd ed. John Wiley and Sons, New York.

246. Schantz, E. J. 1973. Seafood toxicants. In: National Research Council, Food Protection Committee (ed.). Toxicants occurring naturally in foods, 2nd ed. National Academy of Sciences, Washington, D. C.
247. Schaumburg, H. H., R. Byck, R. Gerstl, and J. H. Mashman. 1969. Monosodium L-glutamate: Its pharmacology and role in the Chinese restaurant syndrome. *Sci.* 163:826-828.
248. Schmidt, G. D. and L. S. Roberts. 1977. Foundations of parasitology. C. V. Mosby Co., St. Louis, MO.
249. Seeliger, H. P. R. 1961. Listeriosis. Hafner, New York.
250. Shanbhag, V. V., S. S. Jha, M. S. Kekre, and G. J. Rindani. 1968. Epidemic dropsy in Bombay city and suburbs. *Indian J. Med. Sci.* 22:226-236.
251. Shelokov, A., S. Buckley, J. Casals, J. R. Henderson, W. G. Downs, P. A. Webb, N. H. Wieberga, R. B. Mackenzie, M. L. Kuns, and K. M. Johnson. 1965. Symposium on some aspects of hemorrhagic fevers in the Americas. *Am. J. Trop. Med. Hyg.* 14:789-818.
252. Shewan, J. M. 1962. Food poisoning caused by fish and fishery products. In: G. Borgstrom (ed.). Fish as food, Vol. II. Academic Press, New York.
253. Simon, C. 1966. Nitrite poisoning from spinach. *Lancet* 1:872.
254. Smibert, R. M. 1978. The genus *Campylobacter*. *Ann. Rev. Microbiol.* 32:673-709.
255. Smith, F. A. (ed.). 1966, 1977. Pharmacology of fluorides, Part 1 and Part 2. Springer-Vertag, New York.
256. Smith, H. V., J. M. K. Spalding, and D. M. Oxon. 1959. Outbreak of paralysis in Morocco due to ortho-cresyl phosphate poisoning. *Lancet* 2:1019-1020.
257. Smith, J. W. and K. Wooten. 1978. Anisakis and anisakiasis. *Adv. Parasit.* 16:93-148.
258. Smith, L. D. S. 1977. Botulism: the organism, its toxins, the disease. Charles C. Thomas, Publisher, Springfield, IL.
259. Smith, W. E. and A. M. Smith. 1975. Minamata. Holt, Keenholz, and Winston, New York.
260. Smyth, J. D. 1964. The biology of hydatid organisms. *Adv. Parasitol.* 2:169-219.
261. Snow, J. 1936 (reprinted). Snow on cholera, being a reprint of two papers. Commonwealth Fund, New York.

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CLASSIFICATION OF FOODBORNE DISEASES

